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A CONDITION SUGGESTIVE OF THRESHOLD DENTAL FLUOROSIS OBSERVED IN TRISTAN DA CUNHA

I. Clinical Condition of the Teeth[1]

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INTRODUCTION

The first report on a low incidence of dental caries on Tristan da Cunha by Marshall (1), as well as the subsequent ones by Sampson (2) and Barnes (3), were based on information obtained during only 1 to 2½ day visits to the island. A prolonged study of the islanders had not been made until the author (4) was offered the opportunity to carry out the dental program of the recent Norwegian Scientific Expedition to Tristan da Cunha. During a stay on the island from December 7, 1937 until March 28, 1938, several scientific fields were investigated. The results of the dental study will appear in the near future. This preliminary report will call attention to one previously unreported finding, which, in the light of recent dental research, seems to have a certain significance.

WHITE SPOTS IN THE ENAMEL

Since dental hypoplasia frequently has been associated with resistance to dental caries, we felt that such developmental lesions of the teeth, if present, ought to be carefully recorded. We had, however, little reason to expect such conditions because Marshall mentions that the teeth were "well shaped" and Sampson that they were "strong and well formed, well calcified, and no hypoplasia seen"; while Barnes' records, to which we were given access, list 3 cases of hypoplasia. We were able to verify Barnes' cases as being 3 of the most significant instances of gross hypoplasia. Since these lesions occurred in the incisor

[1] Received for publication January 13, 1941.

TABLE I

The frequency of "white spots" in the enamel of the permanent teeth

TYPE OF TOOTH	NUMBER OF TEETH EXAMINED			NUMBER OF TEETH WITH WHITE SPOTS		
	Upper Jaw	Lower Jaw	Both Jaws	Upper Jaw	Lower Jaw	Both Jaws
1st incisors	284	294	578	100 (35.2%)	23 (7.8%)	123 (21.3%)
2nd incisors	277	290	567	57 (20.6%)	23 (7.9%)	80 (14.1%)
Canines	244	261	505	58 (23.8%)	31 (11.9%)	89 (17.6%)
1st premolars	239	254	493	52 (21.8%)	33 (13.0%)	85 (17.2%)
2nd premolars	231	236	467	36 (15.6%)	27 (11.4%)	63 (13.5%)
1st molars	297	283	580	44 (14.8%)	47 (16.6%)	91 (15.7%)
2nd molars	230	228	458	26 (11.3%)	32 (14.0%)	58 (12.7%)
3rd molars	134	125	259	11 (8.2%)	29 (23.2%)	40 (15.5%)
All teeth	1936	1971	3907	384 (19.8%)	245 (12.4%)	629 (15.8%)

TABLE II

Age distribution of "white spots" in the enamel of upper central incisors

AGE RANGE IN YEARS	NUMBER OF INDIVIDUALS EXAMINED			NUMBER OF UPPER CONTROLS					
				Examined			With "White Spots"		
	Females	Males	In All	Females	Males	All	Females	Males	In All
6–9	10	6	16	20	12	32	14	10	24 (75.0%)
10–14	10	13	23	20	26	46	10	12	22 (47.8%)
15–19	9	13	22	18	26	44	13	13	26 (59.1%)
20–29	10	13	23	20	26	46	5	5	10 (21.7%)
30–39	10	16	26	20	32	52	8	4	12 (23.1%)
40–49	9	7	16	18	14	32	2	0	2 (6.3%)
50–81	8	8	16	16	16	32	2	2	4 (12.5%)
All ages (6–81)	66	76	142*	132	152	284	54	46	100 (35.2%)

* In order to account for the rest of the 188 islanders, all of whom were examined, it should be pointed out that in this table we have excluded 1 girl and 4 boys, 6 years old, and 12 girls and 15 boys below that age, because their permanent incisors were as yet unerupted; furthermore, 2 women and 3 men in the age group 30–39, since their incisor teeth were partly or completely calcified during a stay outside Tristan (their teeth were normal); and finally, we have excluded 4 women and 5 men above 50 years of age, 2 of whom settled down on Tristan as adults, the others having first incisors fractured or lost, whereby a proper diagnosis was prevented.

teeth of 3 individuals over 20 years of age, they must have been present at the first examination of the Tristanites in 1926. Consequently, we

cannot on this basis agree that the frequency of such gross hypoplasia has increased in later years.

However, during our examination we did find a developmental lesion of the teeth which was most apparent in the younger population. This was what we will describe as "white spots in the enamel." Clinically, these lesions manifested themselves as changes in the translucency of the enamel, appearing like opaque areas of a whitish color. In contrast to the hypoplastic defects, which Mellanby (5) attributes to nutritional deficiency (vitamin D) during tooth formation, they had a

TABLE III

The frequency and age distribution of "white spots" in the enamel of deciduous teeth

TYPE OF TOOTH		1–3 YEARS (10 CHILDREN)	3–7 YEARS (15 CHILDREN)	7–12 YEARS (28 CHILDREN)	1–12 YEARS (53 CHILDREN)
1st incisors	Examined	40	54	7	101
	White spots	17 (42.5%)	4 (7.4%)	0	21 (20.8%)
2nd incisors	Examined	40	58	22	120
	White spots	9 (22.5%)	2 (3.4%)	0	11 (9.2%)
Canines	Examined	28	60	87	175
	White spots	4 (14.3%)	2 (3.3%)	2 (2.3%)	8 (4.6%)
1st mol.	Examined	32	60	92	184
	White spots	5 (15.6%)	3 (5.0%)	7 (7.6%)	15 (8.20%)
2nd mol.	Examined	23	60	102	185
	White spots	5 (21.7%)	8 (13.3%)	9 (8.8%)	22 (11.9%)
All teeth	Examined	163	292	310	765
	White spots	40 (24.6%)	19 (6.5%)	18 (5.8%)	77 (10,0%)

smooth surface texture. When dried they appeared more glossy than those posteruptive smooth surface lesions which the author recently has described as "pre-caries" (6, 7). The lesions were slight and diffuse in the deciduous teeth, while in the permanent teeth they were well demarcated from the unaffected portions of the tooth surfaces. In typical cases the white spots occurred in most of the teeth and corresponded with areas of contemporaneous enamel formation. A compilation of all the affected permanent teeth (Table I) shows that the anterior teeth (especially those of the upper jaw) were most frequently affected. To show the age distribution, we have therefore listed

separately in Table II the lesions occurring in the first maxillary incisors.

The table shows that approximately 60 per cent of the maxillary first incisors of the islanders below 20 years of age had evidence of white spots in the enamel, as compared with only 17 per cent in the older population. If the severity of the lesions is taken into consideration, this difference in young and old appears even more striking.

TABLE IV

Number of individuals with "white spots" in the enamel of permanent and deciduous teeth

DENTITION	YEAR OF BIRTH	AGE	NUMBER EXAMINED	MODERATE AND MILD LESIONS	SLIGHT AND QUESTIONABLE LESIONS	NORMAL
Permanent	1918–19	18–19	8	3	3	2
Permanent	1920–21	16–17	8	3	2	3
Permanent	1922–23	14–15	10	3	3	4
Permanent	1924–25	12–13	10	2	5	3
Permanent	1926–27	10–11	9	2	3	4
Permanent	1928–29	8–9	10	4	5	1
Permanent	1930–31	6–7	11	3	4	4
Permanent (in all)	1918–31	6–19	66	20 (30.3%)	25 (37.9%)	21 (31.8%)
Deciduous	1932–33	4–5	6	1	2	3
Deciduous	1934–35	2–3	12	2	5	5
Deciduous	1936–37	½–1	6	1	4	1
Deciduous (in all)	1932–37	½–5	24	4*(16.6%)	11 (45.8%)	9 (37.5%)
Permanent and deciduous (total)	1918–37	½–18	90	24 (26.6%)	36 (40.0%)	30 (33.3%)

* All mild lesions.

Only 1 person over 20 years of age showed well-defined white spots in a number of teeth, while the lesions were obvious enough to be revealed at the first smile of many of the younger inhabitants.

In Table III the deciduous teeth have been compiled. It is seen that the frequency of white spots in the enamel is less than in any type of permanent teeth. In addition, the lesions were all of a mild character that easily may have escaped notice unless our time had permitted a minute examination. Finally, Table IV indicates the individual in-

cidence of these lesions in all the younger islanders born between 1918 and 1937. Again it is seen that the children with deciduous dentition are approximately half as frequently affected (16.6 per cent) as the young individuals with permanent teeth (30.3 per cent in persons between 6 and 19 years).

We should probably have failed to attach any particular significance to the finding of these white spots if it had not been for some cases, for

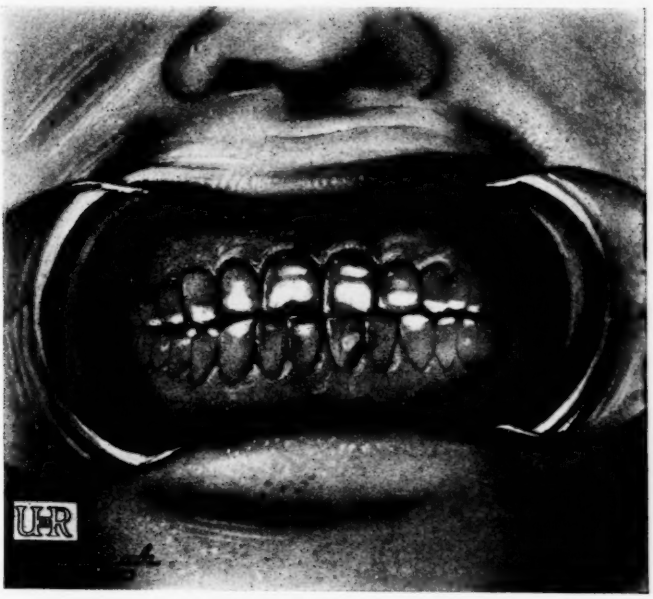

FIG. 1. White spots in the enamel of permanent teeth. (Half tone drawing made by N. C. Jacobs on the basis of photograph, and sketch of an actual case recorded on Tristan da Cunha.)

instance the one in *fig. 1*, which were evident enough to arouse the suspicion of their being a slight degree of dental fluorosis (mottled enamel).

POSSIBLE SOURCES OF FLUORINE

While fluorine in the drinking water generally is accepted as the cause of dental fluorosis in man, food fluorine as well may produce

experimental fluorosis (8). We will consider both the water and food as sources of fluorine in Tristan da Cunha.

The only source of drinking and household water is a spring (*fig. 2*) situated a few hundred meters behind the settlement, just below the steep northwestern side of the 6000 feet mountain cone of the islands, an extinct volcano. From this spring a natural brook (No. 1) runs through the eastern part of the settlement, while 5 artificial brooks, (Nos. 2 to 6) lead the water to the western and newer part of the settle-

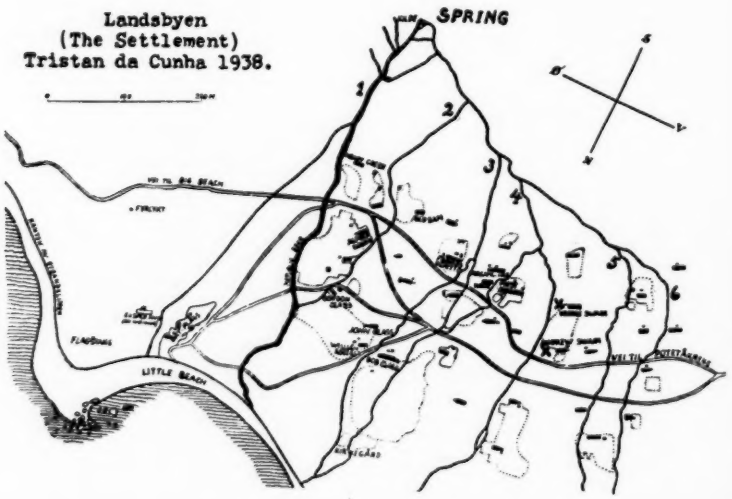

FIG. 2. The water supply of the settlement on Tristan da Cunha. All brooks are coming fom the same spring. No. 1 is the natural brook, while no. 2, 3, 4, 5 and 6 are artificially made. (After map by J. C. Dunne and E. Baardseth 1937/38.)

ment. These latter brooks run slowly, carry little water, and were found to be heavily contaminated with fecal and organic matter. The natural brook ("The big watering") on the other hand, carried excellent water in large quantities. The islanders were well aware of this, and probably this brook has been the principal source of drinking water for most of the inhabitants during the period of tooth calcification. To determine whether the water of this brook contained sufficient fluorine to produce dental changes such as those shown by Dean and Elvove (9) in certain areas of the United States, an analysis was

made of the fluorine and other inorganic constituents. We were fortunate in having the assistance of Dr. H. T. Dean and Chief Chemist Dr. E. Elvove in obtaining an analysis of the Tristan water in the laboratory of the National Institute of Health in Washington, D. C. The results were as follows:

	Parts per Million
Residue on evaporation (103°C.)	275.0
Loss on ignition	17.0
Fixed residue	258.0
Silica (SiO_2)	8.0
Iron (Fe)	0.15
Aluminum (Al)	0.0
Calcium (Ca)	3.2
Magnesium (Mg)	0.8
Sodium and Potassium (calculated as Na)	92.8
Carbonate (CO_3)	33.6
Bicarbonate (HCO_3)	175.6
Sulfate (SO_4)	8.2
Phosphate (PO_4)	0.6
Nitrate (NO_3)	8.3
Chloride (Cl)	10.0
Fluoride (F)	0.2

From this table it may be seen that Tristan's common drinking water is relatively soft, and that its fluorine content (0.2 parts per million) is below the amount (1 part per million) which is considered (9) causative of clinical changes in developing enamel. Even if the fluorine content may vary to some extent in the different brooks and at different seasons, it can hardly explain the fact that mainly children and young people showed clinical evidence of the lesions just described. This led us to consider certain dietary changes as a possible factor.[2]

While potatoes and fish always have been the staple foods of the islanders, the fish foods have become increasingly important to the growing community due to failures of potato crops. In 1938, for example, we found that only 5 out of the 39 Tristan families had the 20–25 bushels of potatoes per individual which the islanders considered sufficient for a year. Three representative families (14 individuals, 9–65 years old), whose food intake was measured during a whole year

[2] This question will be dealt with in detail in the final report which is planned to be published by Det Norske Videnskaps-Akademi, Oslo, Norway.

(1938) consumed respectively 239, 383 and 392 grams of fish per individual per day. Since fish food is one of the richest fluorine sources amongst common natural foods (10), it seems likely that the Tristanites here have a source of fluorine which, when added to the water fluorine, well may explain the lesions resembling dental fluorosis, and as shown elsewhere (11) the high fluorine content of their teeth.

CASE DESCRIPTIONS

The most significant dental lesions occurred in 2 families living at water brook No. 4 (see *fig.* 2, where the houses are marked with crosses).

Family a. In the lower house a family with 10 children grew up. The youngest, a boy of 13, showed one of the most severe cases of "white spots" in the enamel, almost half of the labial surface of some teeth being affected. The children next in age, 19, 25 and 28 years, showed some signs of such lesions, while the teeth of the parents and the 6 older children, with 1 questionable exception, appeared normal.

Family b. In the neighboring house lives the third oldest son of the above family. There are 4 children, 3, 9, 12 and 16 years old. Although a clinical diagnosis of the permanent teeth was possible only in the case of the 2 older children, it could be seen that the erupted first incisors of the 9 year old child were affected. In the 12 year old child, 19 out of 24 erupted permanent teeth had white spots in the enamel, while the 16 year old child showed the most extreme condition on the island, having 25 out of 28 erupted permanent teeth affected. But for these developmental lesions, all the children had perfect teeth, without any traces of caries.[3] Judging by the potato crops, as the islanders do, this family was the poorest on the island and is known to have subsisted mainly on fish. It may be added here that the affected part of the teeth of the 16 year old boy must have been in the process of calcification at a time (1922 to 1925) when severe failures of the potato crops are reported (12) to have occurred.

COMMENTS

For many years the occurrence of dental fluorosis, or mottled enamel, as it was called before, was looked upon as a public health problem. Consequently the field studies were carried out primarily to determine whether the dental lesions were severe enough to necessitate a change of the common water supply containing the causative agent. Such surveys brought out that when the water contained less than 1 part per million of fluorine, the clinical manifestations in the teeth were of no public health significance (9). Thus certain lesions that were less apparent because they lacked in extent and discoloration, were grouped as questionable from a public health viewpoint.

Since the susceptibility to dental caries during the last few years has been related to the fluorine intake (13) and the fluorine content of the

[3] Checked by complete series of radiographs.

teeth (14), the lower fluorine levels and the milder dental lesions immediately gain in significance. Concentrating our attention on the minute changes in the enamel our findings have indicated that the deciduous teeth can present lesions similar to those found in permanent teeth. This does not appear to be in accordance with the general belief that deciduous teeth escape such lesions. As an exception may be mentioned a recent report on a mottled enamel area in India by Day (15) who in a careful examination revealed that mottling in the deciduous teeth occurred approximately half as frequently as in the permanent teeth. This relation is fairly close to what we have observed in Tristan da Cunha, although the absolute incidence and the fluorine content of the water are considerably lower.

On a water supply containing as low as 0.7 parts of fluorine per million, Arnim, Aberle and Pitney (16) observed what they also chose to call "white spots" in the enamel of the permanent teeth in a group of Indian children, 7 to 14 years of age. Similarly to Dean, Jay, Arnold, McClure and Elvove (13) they found that children with such teeth showed a remarkable low incidence of dental caries, particularly in the incisor teeth. Thus, no caries was found in 1.605 incisors, 24 per cent of which had white spots in the enamel. In comparison, our findings in Tristan da Cunha show that out of 1.145 permanent incisors, 17.7 per cent were affected with white spots in the enamel, while no carious lesions were present, except in some of the adult people.

Although our studies of the teeth, water and diet of the Tristanites lead us to believe that we are dealing with a condition of threshold dental fluorosis, we can not disregard the possibility that other causative factors than the ones here discussed may be involved. It is, for example, not clearly understood to what extent the balance of other minerals may influence the production of such lesions, directly or through interaction with fluorine. However, it seems to us that our observations have some general significance because they do suggest that factors besides the water fluorine should be taken into consideration when studying lesions resembling dental fluorosis.

SUMMARY

A condition, not previously reported, suggesting a threshold degree of dental fluorosis has been observed in the inhabitants of the South

Atlantic island of Tristan da Cunha. Out of 3907 permanent teeth, 629 or 15.8 per cent were affected with "white spots" in the enamel, being most frequent in the younger individuals. Out of 765 deciduous teeth, 77 or 10 per cent were affected. The anterior teeth while remarkable free from dental caries, were most frequently affected with white spots in the enamel.

The Norwegian Scientific Expedition to Tristan da Cunha was under the leadership of Dr. Erling Christophersen, Botanical Museum, Oslo, and sponsored by many scientific foundations and private persons in Norway. This being the first report on the dental studies, the author wants to thank all who contributed to the odontological purposes of the expedition. Grants were receivd from Den Norske Tannlaegeforening (Norwegian Dental Association) and A/S Norsk Dental Depot's Foundation. My associates at Norway Dental School, in particular Drs. I. Reichborn-Kjennerud, G. Toverud and J. Rygge, cooperated in preparing the program. Disponent A. Larsen, Advokat E. Onsager, A/S Norrigefa, A/S Nygaard and Company, and A/S Siko offered financial and material aid. I am greatly indebted to A/S Norsk Dental Depot (Disponent A. Larsen), who put at our disposal a complete x-ray outfit, including motor aggregate, which was most valuable to our purposes. The information obtained through Dr. J. A. Moore, Cape Town, and Dr. E. H. V. Barnes, England, who previously have examined the Tristanites, has been very useful.

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A CONDITION SUGGESTIVE OF THRESHOLD DENTAL FLUOROSIS OBSERVED IN TRISTAN DA CUNHA

II. Fluorine Content of the Teeth[1]

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Armstrong and Brekhus (1, 2) have shown that a relatively high fluorine content is characteristic not only for fluorosed teeth but also for sound teeth (without clinical evidence of fluorosis) as compared with carious teeth. Since severe dental fluorosis is as undesirable as dental caries, it is of considerable interest to determine, firstly, the maximum fluorine content of the teeth compatible with their normal or nearly normal appearance, and, secondly, the optimal fluorine content, if such exists, necessary for preservation of sound teeth. Some information on these points may be derived from the fluorine determinations here reported. These analyses were made on a material of deciduous and permanent teeth collected by one of us (R. F. S.) during the recent Norwegian expedition to the South Atlantic island of Tristan Da Cunha (3), where a low incidence of dental caries was observed stimultaneously with a condition resembling threshold dental fluorosis (4).

MATERIAL

The deciduous teeth represent a random sample, so far as they are only from those islanders who were changing dentition at the time of the stay on the island. These teeth were entirely free from caries. With 2 exceptions, they were obtained at the time of normal exfoliation. Permanent teeth were obtained only when malposition or caries justified extraction. Consequently our material is lacking teeth with the characteristic "white spots" in the enamel, previously described

[1] Received for publication January 10, 1941.

(4), since such teeth were always remarkably well preserved, both in the deciduous and permanent dentition. Upon removal the teeth were stored in 70 per cent alcohol, either immediately or following fixation in 8 per cent formalin (in cases where histological sections were desired). Subsequently the teeth were submitted to an extensive study of surface texture, structure, physical and chemical properties,[2] to be reported in detail later. For the fluorine determinations 18 of the teeth were used, 10 deciduous and 8 permanent. Samples of enamel and dentin were obtained from each tooth by powdering and separation, according to the centrifugation-flotation method of Manly and Hodge (5). The samples were analyzed at the University of Minnesota in the laboratory of one of us (W. D. A) who remained unacquainted with the clinical details of the material until completion of the determinations. Analyses were made on each single dentin and enamel sample by the method of Armstrong (6).

RESULTS

In Table I are summarized all the data respective to age and sex of the individuals, the history of the teeth and position of the teeth in the jaws. The total average fluorine content of the enamel was the same for the deciduous and permanent teeth, namely, 0.0140 per cent while the fluorine content of dentin is 0.0196 per cent for deciduous teeth and 0.0270 per cent for permanent teeth. It is observed that the average fluorine content of the dentin of permanent teeth is higher than that of deciduous teeth. The reason for this may be that the dentin samples from the permanent teeth include the whole roots while those from the deciduous teeth include only the crown dentin, the roots being physiologically resorbed. It is conceivable, however, that the permanent teeth with their higher posteruptive age than the deciduous teeth, have been exposed to a greater secondary enrichment with fluorine after their eruption.

The deciduous teeth which we have analyzed, represent quite a unique material, because they had been exposed in the mouths for the complete functional period without being attacked by caries, and were

[2] Carried out at the University of Rochester School of Medicine and Dentistry, Rochester, New York with a grant from the Carnegie Corporation of New York.

TABLE I

Fluorine determinations on teeth from Tristan da Cunha

NAME	SEX	AGE	DECIDUOUS TEETH			FLOURINE (PER CENT)		FLUORINE (PER CENT)	
			Sample	Position	History	Enamel	Average	Dentin	Average
G. L.	M	$7\frac{5}{12}$	4	$\overline{1}\|$	Exfoliated caries-free	0.0137	0.0137	0.0250 0.0259	0.0254
L. L.	M	$9\frac{7}{12}$	9	$\overline{3}\|$	Exfoliated caries-free	0.0140	0.0140	0.0305 0.0294	0.0299
L. L.	M	$9\frac{7}{12}$	10	$\lceil\overline{3}$	Exfoliated caries-free	0.0190?	0.0190?	0.0223 0.0223	0.0223
I. L.	F	$11\frac{8}{12}$	11	$\underline{3}\|$	Exfoliated caries-free	—	—	0.0240 0.0237	0.0238
I. L.	F	$11\frac{8}{12}$	14	$\lceil\overline{4}$	Exfoliated caries-free	0.0134	0.0134	0.0176 0.0160	0.0168
L. L.	M	$9\frac{7}{12}$	15	$\lceil\overline{4}$	Exfoliated caries-free	0.0144	0.0144	0.0152 0.0146	0.0149
A. R.	F	$10\frac{11}{12}$	19	$\overline{5}\|$	Exfoliated caries-free	0.0120 0.0112	0.0116	0.0140 0.0133	0.0139
D. L.	F	$11\frac{11}{12}$	20	$\lceil\overline{5}$	Exfoliated caries-free	0.0106 0.0109	0.0107	0.0128 0.0133	0.0130
O. L.	F	$13\frac{8}{12}$	24	$\overline{5}\|$	Delayed exf. caries-free	0.0145 0.0150 0.0142	0.0145	0.0185 0.0197 0.0202	0.0194
R. R.	M	$18\frac{8}{12}$	25	$\overline{5}\|$	Delayed exf. caries-free	0.0147	0.0147	0.0170 0.0172	0.0171
Total average for deciduous teeth.....							0.0140		0.0196
			PERMANENT TEETH (ALL EXTRACTED)						
L. G.	F	19	27	$\lceil\overline{3}$	Malposition	0.0099 0.0101	0.0100	0.0222 0.0208	0.0215
A. S.	M	74	31	$\lceil\overline{3}$	Malposition	0.0109 0.0108	0.0108	0.0276 0.0280	0.0278

TABLE I—*Concluded*

NAME	SEX	AGE	PERMANENT TEETH (ALL EXTRACTED)			FLUORINE (PER CENT)		FLUORINE (PER CENT)	
			Sample	Position	History	Enamel	Average	Dentin	Average
A. G.	F	$24\frac{6}{12}$	32	$\overline{8}\rfloor$	Caries	0.0130 0.0125	0.0127	0.0294 0.0270 0.0279	0.0281
B. S.	M	$67\frac{8}{12}$	34	$\lceil\overline{8}$	Malposition	0.0207 0.0190 0.0189	0.0195	0.0381 0.0394	0.0385
L. H.	F	$35\frac{1}{12}$	36	$\lvert\overline{7}$	Caries	0.0212 0.0203	0.0207	0.0308 0.0314	0.0311
M. R.	F	$41\frac{4}{12}$	38	$\lvert\overline{7}$	Caries	0.0116 0.0115	0.0115	0.0207 0.0210	0.0208
B. S.	M	$67\frac{4}{12}$	39	$\overline{6}\rfloor$	Caries	0.0107 0.0103	0.0105	0.0250 0.0257	0.0253
A. S.	F	$25\frac{9}{12}$	40	$\lvert\overline{7}$	Caries (hypoplastic)	0.0169 0.0170 0.0170 0.0163	0.0168	0.0234 0.0238	0.0236
Total average for permanent teeth.....							0.0140		0.0270

obtained from entirely caries-free children[3] living in the same environment as members of an isolated community. These facts probably justify some further analyses of the data pertaining to the deciduous teeth.

Most striking is, perhaps, a comparison between the anterior and posterior teeth (Table II), indicating that the anterior teeth have a definitely higher fluorine content than the posterior teeth. In 2 cases where we could compare anterior and posterior teeth from the same mouths, these differences are substantiated, at least as far as the dentin is concerned. As seen from Table I, 2 of the deciduous teeth were several years delayed in exfoliation time. It is interesting to note the high fluorine content both in the enamel and the dentin of these teeth, as compared with normally exfoliated teeth (Table III). The per-

[3] In all cases checked by x-ray diagnosis.

TABLE II

Comparison of fluorine content of anterior and posterior deciduous teeth

Deciduous teeth exfoliated from different mouths

TYPE OF TOOTH	PERCENTAGE FLUORINE			
	Enamel		Dentin	
Incisor	0.0137		0.0254	
Canine	0.0140		0.0299	
Canine	0.0190?		0.0223	
Canine	—		0.0238	
Average		0.0155?		0.0253
1st molar	0.0134		0.0168	
1st molar	0.0144		0.0149	
Average		0.0139		0.0158
2nd molar	0.0116		0.0139	
2nd molar	0.0107		0.0130	
Average		0.0111		0.0134

Deciduous teeth exfoliated from the same mouths

NAME	TYPE OF TOOTH	PERCENTAGE FLUORINE	
		Enamel	Dentin
L. L.	Canine	0.0190?	0 0223
L. L.	1st molar	0.0144	0.0149
I. L.	Canine	?	0.0238
I. L.	1st molar	0.0134	0.0168

TABLE III

Comparison between fluorine content of deciduous teeth with normal and delayed exfoliation

NAME	AGE	TYPE OF TOOTH	HISTORY OF TOOTH	FLUORINE CONTENT PER 100 GM. (PER CENT)			
				Enamel	Average	Dentin	Average
A. R.*	11	Lower 2nd decid. molar	Normal exfoliation	0.0116	0.0111	0.0139	0.0134
D. L.**	11½	Lower 2nd decid. molar	Normal exfoliation	0.0107		0.0130	
O. L.**	13½	Lower 2nd decid. molar	Delayed exfoliation	0.0145	0.0146	0.0194	0.0182
R. R.*	18	Lower 2nd decid. molar	Delayed exfoliation	0.0147		0.0171	

* A. R. and R. R. are sister and brother.

** D. L. and O. L. are cousins (girls).

manent teeth were obtained from individuals of such varying age and of such different histories, that a further analysis is hardly justified. In the only case where we can compare a carious and a normal tooth from the same mouth, the result seems to be in keeping with those previously reported by Armstrong and Brekhus (2). Thus, the sound tooth had a noticeably higher fluorine content than the carious tooth, as seen below.

NAME	AGE, YEARS	TYPE OF TOOTH	PERCENTAGE OF FLUORINE	
			Enamel	Dentin
B. S.	67	Normal sound third molar	0.0195	0.0385
B. S.	67	Carious second molar	0.0105	0.0253

The basis for this comparison is, however, not quite satisfactory, since since these teeth are usually developed at about 10 years' interval.

Some interest was attached to the analysis of 3 carious second permanent molars, extracted from 3 women, 25, 35 and 41 years old. The first one was the oldest unmarried woman on the island, the second was at the time in the latter part of her fourth pregnancy during 5 years, while the third one was the only woman on the island who had been married for many years without giving birth to a child. The values obtained by the fluorine analyses were as follows:

NAME	AGE, YEARS	TOOTH	FLUORINE CONTENT (PER CENT)	
			Enamel	Dentin
A. S.	25	$\overline{7}$	0.0168	0.0236
L. H.	35	$\overline{7}$	0.0207	0.0311
M. R.	41	$\overline{7}$	0.0115	0.0208

It may be surprising that the highest fluorine content was found in the tooth sample from the pregnant woman. It has been recorded that this woman had no caries before she was 30 years old (1932)[4], which seems to indicate a low caries susceptibility. However, at the present examination (3) the attention was immediately drawn to a severe attack of caries affecting several teeth and accompanied by the rare

[4] Personal communication from Dr. H. N. V. Barnes, England.

experience of toothache. It must be left for a later report to discuss the great sexual difference in caries susceptibility of the adult islanders, a condition which obviously is influenced by other factors than the fluorine content of the teeth.

COMMENTS

The literature is unfortunately scanty in fluorine analyses of deciduous teeth. Pointing out the difficulty in obtaining sound deciduous teeth, Armstrong and Brekhus (1) analyzed a pooled enamel sample from the crowns of 3 such teeth whose roots had been physiologically resorbed. The sample contained 0.0072 per cent fluorine. The enamel of the deciduous teeth from Tristan da Cunha has a fluorine content (0.0140) which is nearly twice as high. More analyses have been made on permanent teeth. Thus Armstrong and Brekhus report the important finding that enamel from sound teeth contains 0.0111 per cent fluorine, as compared with 0.0069 per cent in the enamel of carious teeth from persons living in Minnesota. The permanent teeth extracted on Tristan da Cunha contain 0.0140 per cent fluorine in the enamel, which is 50 per cent over the average value given by Armstrong and Brekhus for carious teeth (0.0069) and 27 per cent over that for sound teeth (0.0111). The fluorine content of the dentin is even further above that reported for normal teeth. Thus the average fluorine content of the dentin of permanent teeth (0.0270) is 60 and 71 per cent over that reported by Armstrong and Brekhus (2) for sound (0.0169) and carious (0.0158) teeth, respectively. The dentin therefore has not only an absolutely but also a relatively higher fluorine content than the enamel. That the high fluorine content of the teeth partly may be gained after the eruption is shown by chemical fluorine analyses of rat molars by Armstrong (7) and by Volker, Sognnaes and Bibby (8) who, following systemic administration of radioactive fluorine, were able to trace it in the fully formed teeth of rats and cats.

The high fluorine content of the Tristan teeth is in keeping with the clinical finding of white spots in the enamel, suggestive of threshold dental fluorosis. The high values observed in the deciduous teeth (equal to those in the permanent teeth), may well explain that these teeth presented similar lesions.

The data reported by Sognnaes (4) have indicated that the anterior teeth of the islanders were most frequently affected with white spots in the enamel, and were remarkably free from dental caries. A difference in chemical composition, i.e., in the fluorine content between anterior and posterior teeth, has been observed simultaneously. If confirmed on an equally pure but larger material, this may possibly explain the inverse relationship, which is known to exist, between anterior and posterior teeth with respect to their susceptibility to dental fluorosis on the one hand, and dental caries on the other.

SUMMARY

The average fluorine content of deciduous and permanent teeth from the island of Tristan da Cunha was found to be 0.0140 per cent for the enamel in both dentitions, while the dentin contained 0.0196 and 0.0270 per cent fluorine in the deciduous and permanent teeth, respectively. These values are from 27 to 60 per cent higher than those of non-fluorosed sound teeth (from Minnesota), analyzed by the same method in the same laboratory (1, 2).

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ASSOCIATION OF DENTAL CARIES IN SCHOOL CHILDREN WITH HARDNESS OF COMMUNAL WATER SUPPLIES[1]

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Reports have appeared in Europe (1, 2, 3) and the United States (4) that increased hardness of communal water supply was associated with less dental caries in children. Another investigator in a preliminary report (5) did not find this to be true. We thought it would be worth while, incidental to other studies, to test this relationship in 109 United States cities where we had reliable data of the mineral content of the common water supply and data concerning the incidence of carious permanent teeth of the children of 3 age groups residing in those cities. The dental data were collected under the direction of and published by the U. S. Public Health Service (6). The "hardness," as $CaOO_3$, data were furnished by the United States Department of Interior, Geological Survey (7).[2] The method of computing the number of various permanent teeth per 100 children has been described elsewhere (8).

The mean number of carious permanent teeth for the 109 cities, arranged according to age are shown in Table I.

The frequency distributions of the 109 cities, based on the number of carious permanent teeth per 100 children are shown in *fig. 1*, plotted on probability paper. The various values closely follow the normal curve of error and are, therefore, thought to be suitable for comparing the number of affected permanent teeth of one group with another and for the purpose of determining the degree of relationship between carious teeth and the hardness of the common water supplies.

The degree of association of the "total hardness as $CaCO_3$" of the

[1] Received for Publication January 24, 1941.

[2] The values for hardness are those which pertain to the water *as it reaches the consumer*. In some cities it is the practice to "soften" the water for industrial and domestic use.

TABLE I

Mean number of carious permanent teeth according to age (109 cities)

NUMBER OF CITIES	AGE GROUP	MEAN NUMBER CARIOUS PERMANENT TEETH PER 100 CHILDREN
109	6–8	113.5 ± 3.6*
	9–11	275.0 ± 6.0*
	12–14	449.0 ±20.2*

*$\sigma m = \frac{\sigma}{\sqrt{N}}$.

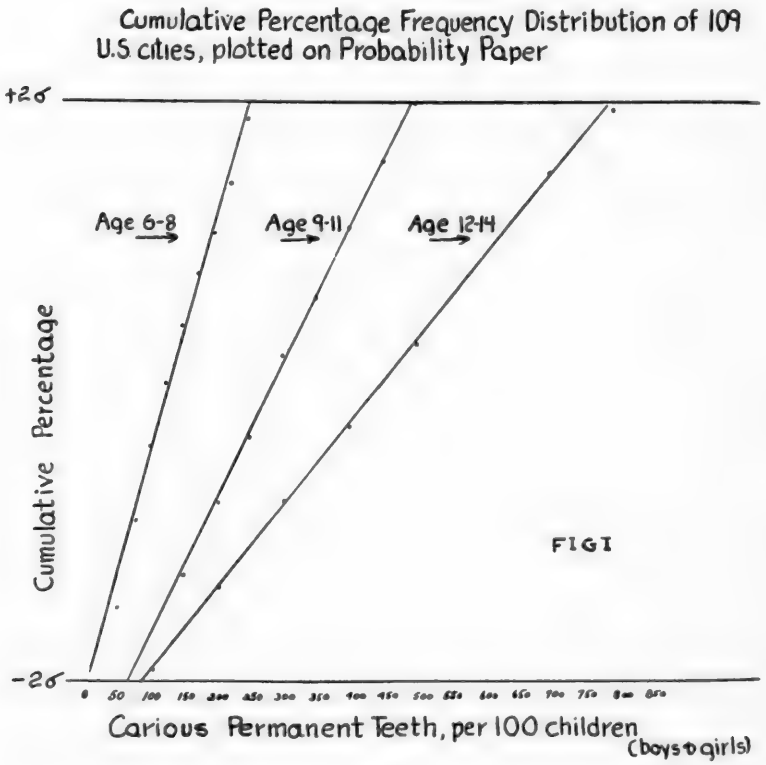
Cumulative Percentage Frequency Distribution of 109 U.S cities, plotted on Probability Paper
+2σ
Age 6-8
Age 9-11
Age 12-14
Cumulative Percentage
FIG I
-2σ
0 50 100 150 200 250 300 350 400 450 500 550 600 650 700 750 800 850
Carious Permanent Teeth, per 100 children
(boys & girls)

FIG. 1

communal water supplies and the mean number of permanent teeth which had been attacked by caries was determined by the correlation

coefficient. The coefficients for the various age groups of children, boys and girls combined, are given in Table II. Negative relationships exist between high hardness and high caries in every case. The relation of the respective values to their standard errors demonstrates their statistical significance. The chances are less than 1 to 13,000 that the values are due to random sampling.

TABLE II

Coefficients of various age groups

Age	6–8	9–11	12–14
r*	−.40	−.38	−.41
σr**	.09	.09	.09
x/σ	4.4	4.2	4.5

$$^{*}r = \frac{\frac{\sum dxdy - cxcy}{N}}{\sigma x \sigma y}.$$

$$^{**}\sigma r = \frac{1 - r^2}{\sqrt{n}}.$$

TABLE III

Correlation of water hardness and mean annual hours of sunshine and latitude

Number of cities	Mean annual hours of sunshine	Mean hardness of water as $CaCO_3$ (ppm)	Mean latitude
109	2596.0 ±22.1	136.8 ±9.7	41.1 ±2.3
r	(low sunshine—low water hardness) .12 ±.09		−.02 ±.09 (low hardness — low latitude)
x/σ	1.3		0.2

Elsewhere we (8, 9, 10) have demonstrated the high degrees of association of high caries with those environmental factors which decrease the amount of vitamin D producing solar energy at the place of residence of children. The 109 cities which are the basis of this study constitute a part of the locations included in the environmental studies. Therefore, it is important to determine the overlapping influence of the 2 climatic factors which were found to be important in the previous studies, mean annual hours of sunshine and latitude.

If it can be shown that high hardness of water is in sufficient positive correlation with mean annual hours of sunshine or in sufficient negative correlation with latitude, we would be inclined to conclude that the negative relationship between high caries and high hardness is apt to be accidental. The correlation of "total hardness, as $CaCO_3$" of the water supplies of the 109 cities and the mean annual hours of sunshine and the latitude of those cities was determined. The values are given in Table III.

There appears to be no significant relationship between either mean annual hours of sunshine, latitude and the mean hardness of the 109 cities in our series. Therefore, it seems proper to suggest that the negative relationship of high hardness of the communal water supplies and the high caries in these cities is a factor in caries incidence which should be given consideration in its own right. It would not be surprising, or contrary to experience in calcium and vitamin D metabolism, if this were true; in fact it is what should be expected.

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GUTTAPERCHA CONES AS BACTERIOSTATIC AGENTS[1]

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Guttapercha, due to its chemical and physical properties, cannot be heat sterilized. The degree of heat required for sterilization would distort the shape of the cone and also cause adherence of one cone to another. Further, once a box of guttapercha cones is opened, the cones become accessible to potential contamination either by microorganisms present in the air or by contacting objects. On the other hand, since the guttapercha cone is dry and supposedly free of bacterial pabulum its surface may be compared to that of an unused metallic instrument. The status of the guttapercha cone, therefore, from the standpoint of sterility was considered of sufficient interest to warrant this bacteriological investigation.

Experiment 1. The initial test consisted of inserting individual cones into tubes of infusion broth. Twelve cones of each of the 3 principal manufacturers, i.e., White, Caulk and Mynol, were examined. No growth of bacteria was evident in any of the tubes after an incubation period of 3 days. Half the tubes were kept at room temperature while the rest were incubated at body temperature. Since the cones were of different shades of pink it was thought possible that some substance such as a dye was present and acted as a bacteriostatic agent.

Experiment 2. Tubes of infusion broth, pH 7.8 (volume 2.5 to 3 c.c. per tube) were inoculated with a suspension of *Staphylococcus aureus*. The inoculum contained approximately 100,000 organisms as estimated by a colony count of a poured plate. Guttapercha points of the 3 manufacturers were placed in these broth tubes. As controls one of the inoculated tubes received no cone, while to another, a small piece of white base plate guttapercha was added. The experiment was performed in duplicate. No observable growth was noted in the

[1] Read at a Faculty Staff Meeting, School of Dental and Oral Surgery, Columbia University, December 11, 1940. (Received for publication Jan. 15, 1941.)

2 tubes containing the Caulk guttapercha cones during a period of 7 days while the other tubes of broth were cloudy in 24 hours.

Experiment 3. Duplicate tubes of broth were inoculated respectively with suspensions of *Staphylococcus aureus*, *B. coli* and *B. subtilis*, the latter a spore bearing organism. Inhibition of growth occurred only in the *Staphylococcus aureus* and *B. subtilis* inoculated tubes to which the Caulk cones were added. During a period of 1 week's incubation no growth was apparent in these 4 tubes. The gram negative *B.*

TABLE I

Tests for bacteriostatic activity of guttapercha cones in broth

TEST MICROORGANISMS	CAULK*		WHITE*		MYNOL*	
	No. Cones Tested	Results	No. Cones Tested	Results	No. Cones Tested	Results
Gram positive						
Staphylococcus aureus**	10	No growth	10	Growth	10	Growth
B. subtilis**	8	No growth	8	Growth	8	Growth
Pneumococcus Type I	6	No growth	6	Growth	6	Growth
Hem. streptococcus	6	No growth	6	Growth	6	Growth
Strept. viridans	6	No growth	6	Growth	6	Growth
Gram negative						
B. coli	8	Growth	8	Growth	8	Growth
B. typhosus	6	Growth	6	Growth	6	Growth
B. pyocyaneus	6	Growth	6	Growth	6	Growth

* Cones from 2 other lots of each manufacturer's product were tested. No bacteriostatic activity was evident with these cones of each manufacturer.

** *B. subtilis* and *Staph. aureus* were dried on 20 cones of each manufacturer. At 24 hour intervals they were placed in broth. No growth of organisms occurred with the Caulk cones, whereas even after drying 5 days, growth was evident with the other manufacturers cones.

coli grew profusely within 24 hours. The restraint of growth of the 2 gram positive organisms was shown to be bacteriostatic since loopfuls of the clear broth from the tubes containing these organisms were streaked on blood agar plates and pure cultures of both the *Staphylococcus aureus* and the *B. subtilis* developed. The above test was repeated 3 separate times with the same results.

Experiment 4. The guttapercha cones of the 3 manufacturers were now tested for bacteriostatic activity against additional bacteria. Du-

plicate tubes of broth were inoculated with suspensions of such gram positive organisms as the *Pneumococcus, type I*, a hemolytic streptococcus, a streptococcus of the viridans group, and 2 gram negative organisms, *B. pyocyaneus* and *B. typhosus*. These tests were also repeated 3 times each. It was found that the gram positive organisms were inhibited in their growth by this one type of guttapercha cone while the gram negative bacilli were not restrained.

Experiment 5. Twenty guttapercha cones of each manufacturer were now tested by immersing them in heavy saline suspensions of *Staphylococcus aureus* and *B. subtilis*, respectively. After an immersion of 30 minutes, the cones were removed to sterile petri dishes and allowed to dry for 24 hours. Then, at 24 hour intervals, some of the cones covered with the dried organisms were placed in tubes of broth. No growth occurred in the broth tubes to which the Caulk cones containing organisms dried 24 or more hours were added. Growth of the inoculating organisms occurred in the broth tubes in which the White and the Mynol cones were placed even though the organisms on them had remained in a dry state for 5 days.

Experiment 6. Inquiries of the manufacturers as to what material was used to color their guttapercha cones elicited the following information: Caulk's points contained red iron oxide (0.6 per cent); White's points mercuric oxide; Mynol points carmine. Red iron oxide was now added to an infusion broth to give a concentration of 0.6 per cent, an amount obviously much more than the iron oxide content of a single guttapercha point. Heavy growths of *B. subtilis* or *Staphylococcus aureus* developed in this medium within 24 hours after inoculation.

Experiment 7. Cones from 2 other lots of each manufacturer were tested as outlined above. No bacteriostatic activity could be demonstrated even with the Caulk cones.

Experiment 8. A solid medium containing 10 per cent serum in infusion agar which was seeded heavily with a broth culture of *Staphylococcus aureus* just prior to pouring the medium into plates was also employed. Guttapercha cones were laid on the surface of 3 plates of this medium. A clear zone surrounding the Caulk cone from the first lot indicated inhibition of bacterial growth while the White and Mynol points did not restrain bacterial proliferation.

CONCLUSION

Guttapercha cones are relatively free of organisms according to these experiments. This seems to be due to their dryness and lack of pabulum. One lot of Caulk's guttapercha cones was found to possess a bacteriostatic action towards certain gram positive microorganisms but this characteristic was not due to the iron content of these cones and other lots of the same manufacturer's cones did not possess this property. This bacteriostatic action possibly is due to some constituent of the guttapercha which is variable in quantity.

HEAT TREATMENT OF SOME HIGH-PALLADIUM ALLOYS[1]

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The 3 clasp wire alloys shown in *fig. 1* display interesting differences in their reactions to heat treatment. Alloy "A" is more or less typical of the conventional high-gold, low-platinum-palladium clasp wires, while Alloys "B" and "C" are of the newer high-palladium low-gold types.

In each case, the lower curve represents the tensile strength of samples annealed 5 minutes at the indicated temperature and quenched. The upper curve represents the tensile strength of samples annealed 5 minutes at the indicated temperature and quenched and then hardened by furnace cooling. Alloy "A" was furnace-cooled from 880° to 480° F. over a period of 15 minutes. Alloys "B" and "C" were furnace-cooled from 1000° to 500° F. over a period of 30 minutes.

In the case of Alloys "A" and "B", the tensile strength of the annealed and quenched samples decreased as the annealing temperature increased. In contrast to this, Alloy "C" shows an increase in tensile strength with increase in annealing temperature from 1400° to 1600° F.

In the case of the oven-cooled samples, Alloy "A" shows a decrease in strength as the preliminary annealing temperature increased. Alloys "B" and "C" attained maximum strength with an annealing temperature of 1600° F. and 1450° F., respectively. In the case of Alloy "C", at least, the maximum is not sharp but extends from 1450° to 1550° F. In the case of both of these alloys, the strength curve drops rapidly from the maximum for both higher and lower annealing temperatures.

[1] Paper read at the First Annual Meeting of the Dental Materials Group of the International Association for Dental Research, Philadelphia, Pa., March 13, 1940. (Received for publication March 8, 1941.)

[2] Research Department, The J. M. Ney Company, Hartford, Connecticut.

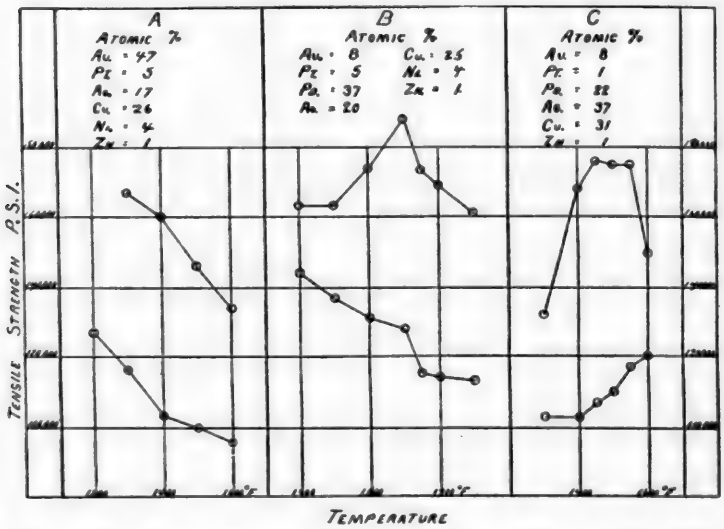

FIG. 1.—Effect of annealing temperature on tensile strength of annealed and of oven cooled wires

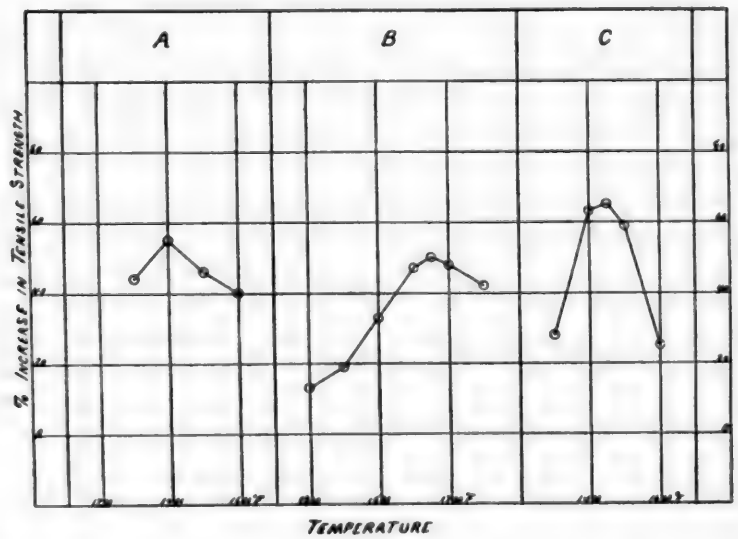

FIG. 2—Effect of annealing temperature on increase in tensile strength produced by subsequent oven cooling

Fig. 2 shows the percentage increase in tensile strength produced by oven cooling for each of the preliminary annealing temperatures. Each of the curves shows a maximum, though for Alloy "A" it is not very pronounced and may be of doubtful significance. In the case of both Alloy "B" and Alloy "C", the effect of the annealing temperature on the percentage increase in strength is very pronounced.

The peculiarities in reaction to heat treatment displayed by Alloys "B" and "C" are characteristic of a number of the high-palladium low-gold alloys in current use and have an important bearing on their handling in the dental laboratory. Some, but not all, of these alloys are made harder in the annealed condition by annealing at higher temperatures, so that in order to obtain maximum softness, the correct annealing temperature must be known and used. In order to obtain the maximum strength in the hardened condition, preliminary quenching from a temperature higher than the 1292° F. specified in the A.D.A. Specification No. 7 for Dental Wrought Gold Wire Alloys is necessary. As a consequence of this characteristic, wires of these alloys allowed to cool slowly in investment from soldering temperatures, will not develop their maximum strength during such slow cooling, nor during a subsequent low temperature aging unless it is preceded by quenching from a relatively high temperature. Also, these alloys are not susceptible to accidental "over-hardening" or embrittling by very slow cooling from soldering or casting temperatures.

GROWTH IN THE MANDIBULAR DENTAL ARCH[1]

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In a previous study on the growth of the dental arches in the cat (1), the rate of percentage growth increase in anterior-posterior and lateral dimensions was found to vary. The direction of growth also was shown to be accelerated during certain periods of development. Because the dentition of the cat follows the human curve so closely (2) (3), and because it is accepted that the growth of the dental arch may be taken as an indication of growth in the jaws, it was thought that a similar study made upon the human dental arch might show comparable findings.

In this study, the dental arches of 544 individuals between the ages of 5 and 25 were measured in 3 directions. All measurements were made upon the mandible from plaster casts of the dentition of children and young adults, and on living subjects. The following points were selected for measurement (*fig. 1*). Lines A and B represent the anterior-posterior dimension from the median line, immediately anterior to the incisor teeth, to the distal gingival margins of the last molar teeth (Dimensions A and B are represented by a single line as they are identical). Line C represents the dimension across the mandible, using the distal gingival margins of the canine teeth as points of measurement. Line D represents the dimension across the mandible in the region of the last molar teeth, using the distal gingival margins as measuring points.

Table I is a compilation of the growth measurements described above and includes a calculation of the percentage growth based on maturity and the earliest age dimensions studied.

[1] Senior students Norman Dimond and Maurice E. Low of the School of Dental and Oral Surgery, Columbia University, made the measurements for this study as part of their thesis requirements. (Received for publication March 17, 1941.)

It will be seen from examination of the graph based on maturity dimensions (*fig. 2*), that A-B, the anterior-posterior dimension represented on the graph by a solid line, increases 15.4 per cent of its total growth between the time of the eruption of the deciduous dentition and that of the first permanent molar tooth. During that same period,

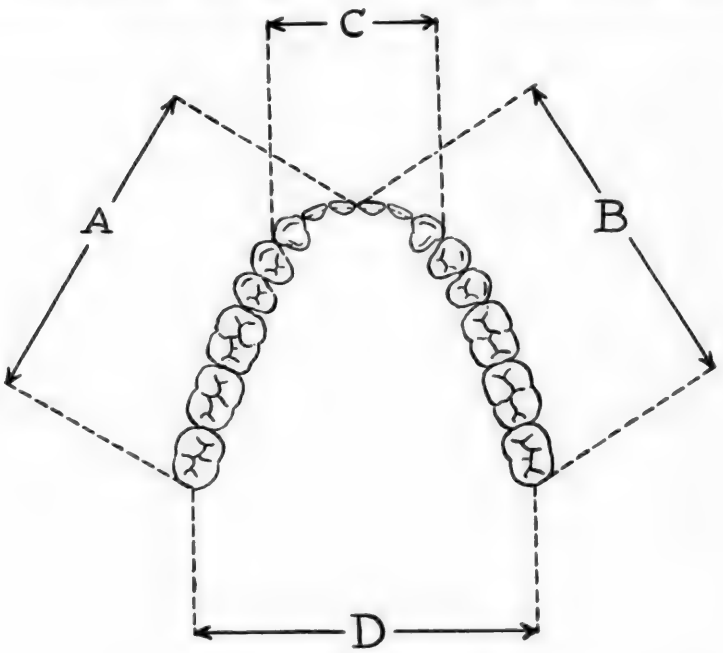

DIAGRAM INDICATING
LINES OF MEASUREMENT
-MANDIBLE-

FIG. 1

D, the lateral dimension between the molar teeth, represented on the graph by a dotted line, increases 9.3 per cent of its total, while C, the interrupted line on the graph representing the lateral dimension between the canine teeth, increases but 5.3 per cent of its maximum.

In the next period, between the ages of 7 and 13, corresponding to the period of the mixed dentition of the child, an interesting change

in the acceleration of the rate of growth between the various dimensions occurs. The anterior-posterior dimension A-B increases only 2.4 per cent, while C, the dimension across the mandible between the canine teeth, grows 5.6 per cent, and D, the lateral dimension between the molar teeth increases 11.4 per cent of its maximum.

TABLE I

AGE	MEASUREMENTS AT THE END OF EACH PERIOD (DIMENSIONS IN MM.)			TOTAL GROWTH GAINED AT THE END OF EACH PERIOD (DIMENSIONS IN MM.)			PER CENT OF MATURITY ATTAINED AT THE END OF EACH PERIOD			PER CENT OF EARLIEST DIMENSION GAINED AT THE END OF EACH PERIOD		
	A-B	C	D	A-B	C	D	A-B	C	D	A-B	C	D
After the eruption of the deciduous dentition ... At age 5	31.4	27.9	36.1	—	—	—	54.2	86.9	65.3	—	—	—
After the eruption of the first permanent molar. At age 7	40.3	29.6	41.4	8.9	1.7	5.3	69.6	92.2	75.0	28.4	6.3	14.4
Previous to the eruption of the second permanent molar..... At age 13	41.7	31.4	46.4	10.3	3.5	10.3	72.0	97.8	86.4	32.8	12.6	28.5
After the eruption of the second permanent molar..... At age 17	49.6	32.1	48.9	18.2	4.2	12.8	85.7	100.0	88.6	58.0	15.1	35.5
After the eruption of the third permanent molar..... At age 25	57.9	32.1	55.2	26.5	4.2	19.1	100.0	100.0	100.0	84.3	15.1	52.9

Between the ages of 13 and 17, following the eruption of the second permanent molar tooth, the anterior-posterior dimension A-B again becomes accelerated in its total percentage growth relative to the other two dimensions. A-B increases 13.7 per cent, while C and D, the dimensions across the mandible in the region of the canines and molars respectively, each grow but 2.2 per cent of their maximum. It is at the end of this period that the lateral canine dimension C, reaches its total percentage growth.

In the final period, between the ages of 17 and 25, after the eruption of the third permanent molar tooth, the anterior-posterior dimension A-B increases 24.3 per cent to attain its maturity growth, while the dimension D, across the molar teeth, grows 11.4 per cent, also attaining its total growth percentage.

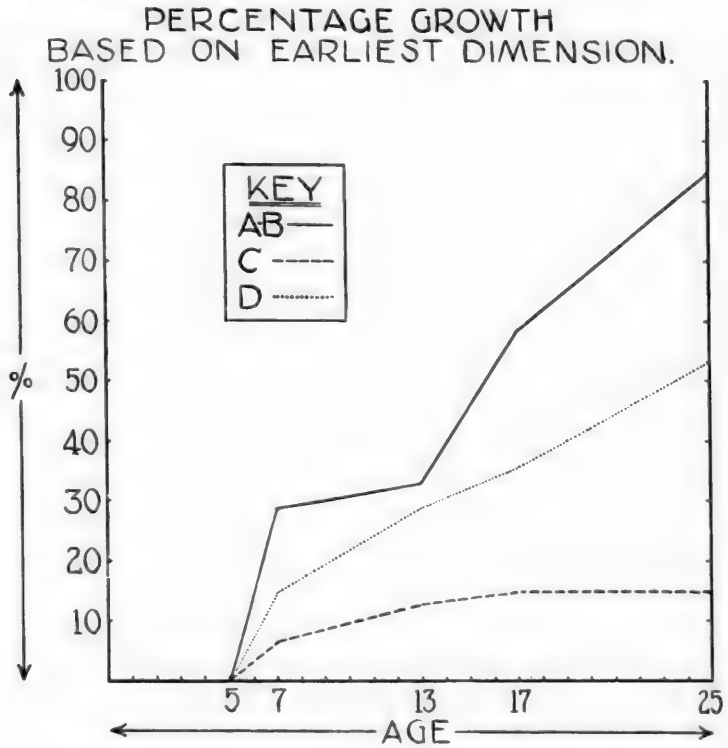

FIG. 2

In the graph based on the earliest age studied (*fig. 3*), a comparable variation in the rate of percentage growth increase is evident.

Table II summarizes the observations described above.

CONCLUSION

1. As in the previous study on the dental arches of the cat (1), this study in the human dental arch shows that the dimensional growth values vary during different periods of development.

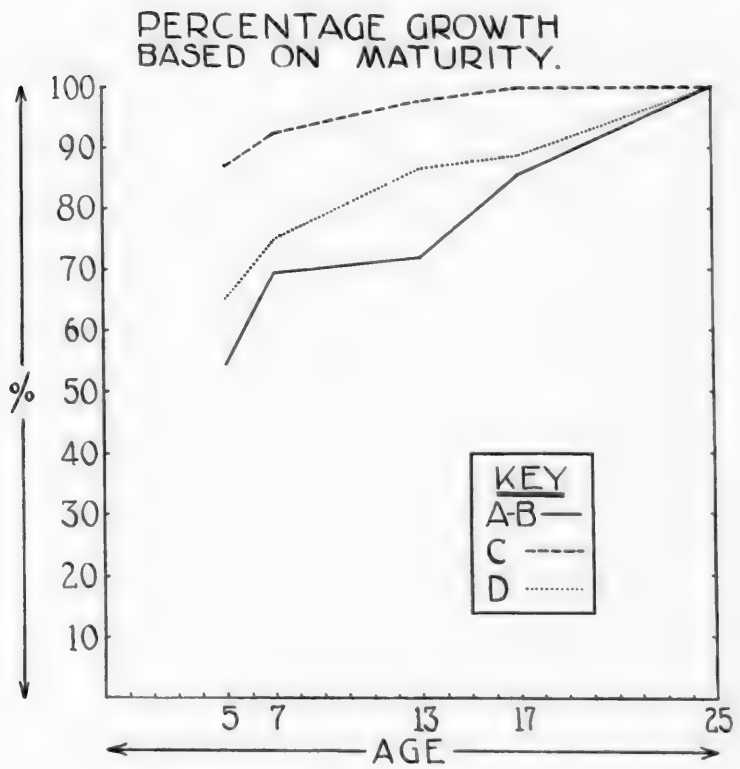

FIG. 3

TABLE II

DIMENSION	PER CENT OF MATURITY DIMENSION (MATURITY = 100%)					PER CENT OF EARLIEST DIMENSION (EARLIEST DIMENSION = 0%)			
	At age 5	Increase between age 5 and ages				At age 7	Increase between age 7 and ages		
		7	13	17	25		13	17	25
A-B	54.2	15.4	17.8	31.5	55.8	28.4	4.4	29.6	55.9
C	86.9	5.3	10.9	13.1	13.1	6.3	6.3	8.8	8.8
D	65.3	9.7	21.1	23.3	34.7	14.4	14.1	21.1	38.5

2. From the study of measurements made on 544 individuals, there seems to be an indication of a difference between the sexes in the rate

of growth of each dimension at different periods. Additional data are needed to verify this observation.

3. These findings may be of interest to orthodontists, who, in inducing growth in the arches, may work with rather than against the natural growth tendencies during certain periods of development.

The writer wishes to express appreciation for technical assistance rendered by Mr. Sidney Shapiro.

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DOES THE MAXILLA LACK A PERIOSTEAL MEMBRANE?[1]

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The periosteum has been a point of contention for almost 200 years, dating from the controversy between Duhamel and Haller (1) concerning its osteogenic or bone-forming power. It is not our purpose to enter into this age old conflict but rather to attempt to clarify the conception which occasionally appears in the literature to the effect that the maxilla lacks, or is poorly provided with, a periosteal covering. Thus, for example, Foman (2) makes the statement that

"since involucra are dependent for their formation upon periosteum, they are scantily formed in the maxilla, due to the poor development of the membrane in this location."

According to Wilensky (3, 4)

"new bone formation here (in the jaws) as in other bones of the body, is dependent to the largest extent upon the presence and the anatomical structure of the periosteum. Inasmuch as in both the upper and the lower jaw there is no true marrow cavity, any new bone formation derived from any endosteal osteoblastic cells is lacking.

"A small area on the anterior surface of the superior maxilla just below and in front of the malar process has a covering that approaches in structure that of a periosteal membrane. The membrane is continuous over the inferior orbital margin with the periosteum covering the floor of the orbit. The palatal surface and the alveolar process are covered with a thick mucoperiosteal membrane. The other surfaces of the bone are covered with the relatively dense attachment of muscles and tendons that find anchorage deep in the bone tissue, and in this respect the structure resembles that of the vertebrae. No periosteal membrane is present on these latter surfaces. . . .

"A periosteal network is practically nonexistent; the little that corresponds to this is derived from an abundant network in the mucous membrane covering the alveolar process of the bone. The physiological proof of this deficiency is found in the total absence of any new bone formation after disease or destruction of any part of the bone."

Before attempting to challenge this concept it is important at the outset to define the term periosteum, since indefiniteness of terminol-

[1] Read at the 19th general meeting of the International Association for Dental Research, St. Louis, Mo., March 15 and 16, 1941 (J. D. Res. 20: 233, 1941) (Received for publication, March 16, 1941).

ogy undoubtedly accounts for a great deal of the confusion relative to the whole aspect of the problem of periosteum. Most histologists are agreed that periosteum, as the term implies, is the membrane which invests bones except at their articular surfaces. To quote Maximow (5) "the modified connective tissue covering of a bone is called periosteum." As Ely (6) pointed out "periosteum is the tissue which covers bone in all situations not covered by the articular cartilage. As with the marrow, the situation of this tissue determines its name, not its structure." As to its structure, it is generally agreed that it consists mostly of fibrous connective tissue in which is a vascular network, the branches of which penetrate via Volkmann's canals into the underlying bone. The question raised by Ham (7) as to whether these cells are osteoblasts or osteogenic, that is, bone-producing cells, and various authors' opinions as to whether or not these cells are a necessary constituent of adult periosteum need not concern us in the present problem. In short, regardless of its structure, mode of attachment, or function of its component cells, concerning which most of the controversy has raged, it is self evident that the periosteum is a connective tissue covering of bone. Moreover the opinion generally prevails that all bones are covered everywhere with this membrane except at their articular surfaces.

Ely (8) makes the following terse and significant remark: "Its (i.e. periosteum) structure has been variously described. The question can be determined not by animal experimentation or clinical phenomena, but only by looking at the tissue under the microscope." The logical method of approach to the problem concerning whether or not the maxilla is everywhere provided with periosteum is to look for such a covering.

The accompanying illustrations show what is found when the maxilla of the human cadaver is examined grossly and microscopically. For comparison illustrations of microscopic sections of the mandible are also shown, since the presence of periosteum on the latter has not been questioned. *Fig. 1* shows an anterior or facial view of a gross dissection of the maxillary region of an adult human cadaver. The periosteum has been detached and reflected from the frontal and zygomatic processes and also from the anterior (facial) and posterior (infratemporal) surfaces of the body of the maxilla. *Fig. 2* shows a

thin (20 micra) cross-section of a decalcified, human maxilla through the molar area. *Fig. 3* is a high power magnification of the area

FIG. 1. Gross dissection of human maxillary region, left side. The periosteum has been lifted away from the surface of the maxilla. *O*—orbit, *F*—frontal process (nasal articulation), *Z*—zygoma.

FIG. 2. Thin cross section through decalcified maxillary region in molar area. Area (*A*) is shown under high magnification in *fig. 3*.

FIG. 3. High magnification of area *A*, *fig. 2*. Note definite cellular membrane (*P*) with osteoblasts arranged along bone (*B*) surface. Presence of vascular network in this membrane is shown by cross sections of several blood vessels (*V*).

marked A in *fig. 2* showing a definite connective tissue membrane in which are cells (osteoblasts?) and a vascular network characteristic of periosteum. *Fig. 4* is a thin cross-section of the mandible, from the

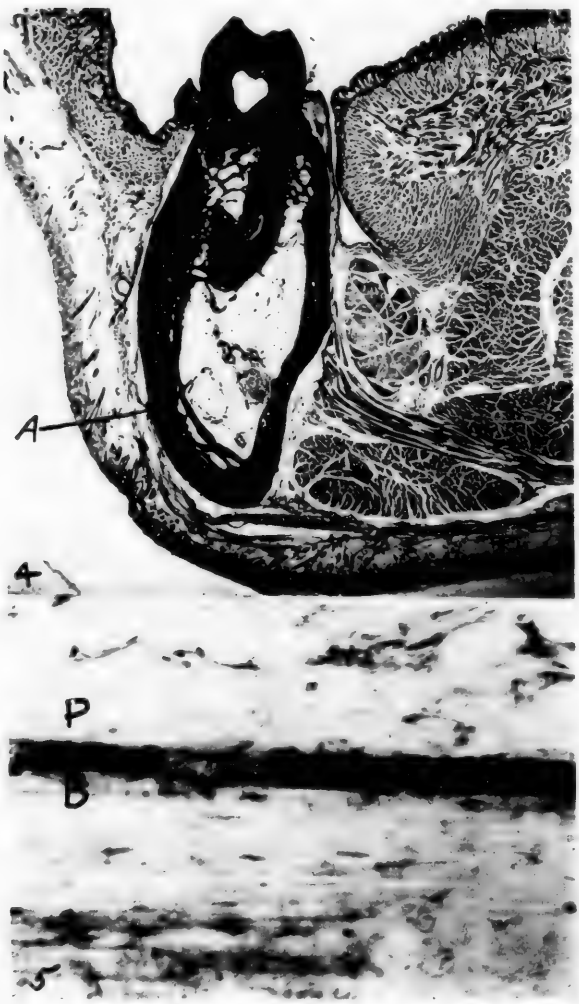

FIG. 4. Cross section of mandible, from same cadaver, in molar region. High power magnification of area marked (*A*) is shown in *fig. 5*.

FIG. 5. High power magnification of area (*A*), *fig. 4*. Fibrous periosteum (*P*) of mandibular bone (*B*) shows markedly fewer osteoblasts than in case of maxilla. Vascular network is much less prominent than in maxillary periosteum.

same cadaver, furnishing the maxillary section, through the molar region. *Fig. 5* is a high power magnification of area A in *fig. 4* showing, as in the case of the maxilla, a definite connective tissue membrane. Although the presence of a periosteum surrounding the mandible has not been questioned, this illustration, which is typical of other sections of the mandible, paradoxically reveals fewer osteoblasts and a less marked vascular network in the periosteum than are shown in that of the maxilla.

In conclusion these illustrations of human maxillae furnish undoubted evidence that this bone is provided with a connective tissue covering which, according to generally accepted definition, is periosteum. We do not dispute the clinical phenomenon of the frequent failure of bone regeneration following extensive necrosis of the maxilla, as in osteomyelitis. However we do challenge the postulation that this clinical phenomenon is explainable on the basis of a lack of maxillary periosteum.

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THE DISTRIBUTION OF PHOSPHATASE IN GINGIVAL TISSUE[1]

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Phosphatase has been demonstrated by chemical methods in gingival tissue and saliva With the chemical methods of phosphatase determination employed so far it is impossible to determine the exact localization of the enzyme in various cells or structures. Tissues are not homogeneous and the enzyme activities of various types of cells may vary considerably. Thus, in gingival tissue which consists of epithelial cells, collagenous fibers, connective tissue cells, intercellular substances, capillaries, arterioles and venules as well as lymph vessels and nerves, phosphatase may be present in some, but not necessarily in all constituents. It is, therefore, worthwhile to study the exact distribution of the enzyme in gingival tissue by a combination of chemical and histological methods.

LITERATURE

Excellent reviews of the literature on phosphatase have been made by Oppenheimer (1) and by Kay (2). The most widely accepted theory of the role of phosphatase in the deposition and maintenance of the mineral salts present in bones and teeth is that of Robison (3). The enzyme is present in bone and ossifying cartilage and is capable of hydrolyzing phosphoric esters which are brought to these tissues by the blood stream. This causes a localized increase in the concentration of inorganic phosphate ions. The deposition of phosphate salts which then follows is regulated by the law of mass action. Robison's theory explains one important factor in the deposition of mineral salts. It does not furnish a complete explanation of all factors involved in these complex processes.

[1] This study has been aided in part by a grant from the Xttrium Company. (Received for publication March 16, 1941.)

Adamson (4) found gingival tissue to contain phosphatase which he believed was capable of hydrolyzing phosphoric esters of the blood stream and saliva.

Smith (5) estimated the phosphatase content of saliva by measuring the amount of hydrolized glycerophosphate. In order to determine whether the enzyme is present in the cellular or in the fluid constituent of saliva, he centrifuged human saliva and examined the supernatant fluid, which he found to be devoid of phosphatase activity. Since the process of centrifuging had removed the enzyme from the saliva, Smith concluded that it must be present in the desquamated epithelial cells. In order to eliminate the action of bacteria in his experiments, Smith incubated saliva after adding chloroform. Destruction of bacteria did not change the phosphatase content of the saliva. He observed that brushing of the gingivae reduces the phosphatase activity, and further, that the presence of inflammation, with increased desquamation augmented the phosphatase activity. Smith interpreted these findings as proofs of his theory that the desquamated epithelial cells contain the phosphatase.

METHODS

Tissues for phosphatase determination were obtained by biopsy from the gingivae of 20 patients whose ages ranged from 12 to 54 years. Five patients had clinically normal gingival tissue. Fifteen patients suffered from gingival disturbances varying from mild inflammation to severe inflammation with marked edema.

The method[2] for the microtechnical demonstration of phosphatase

[2] 1. Biopsy sections of gum tissue are fixed in 95 per cent alcohol.
2. Dehydrated in absolute alcohol.
3. Placed into cedar oil for 2½ hours.
4. Into benzene for one hour during which the solution is changed once.
5. Embedded in liquid paraffin (55°C.) for 2 hours.
6. Blocked and cut into sections of from 5 to 6 microns thick.
7. Placed into xylol until all paraffin is dissolved.
8. Into 95 per cent alcohol for approximately 10 minutes.
9. The sections are dipped into a celloidin solution in order to provide them with a thin coating of celloidin.
10. Dried in air for 1 minute.
11. Into 95 per cent alcohol, 75 per cent alcohol and water for a few minutes each. The sections are now ready to be used for the demonstration of phosphatase. They are

in tissue was first described by Gomori (6) in 1939. It is based upon the following facts:

1. Phosphatase is not immediately destroyed by alcohol (90 per cent or 75 per cent). If dehydrated completely (by alcohol) it withstands heating to 55°C. for 2 hours. The procedures used for the fixation of gingival tissue and its embedding into paraffin or cellodin, therefore, do not harm the phosphatase contained there.

2. When tissue containing active phosphatase is incubated with a solution of sodium glycerophosphate PO_4 ions will be split off. This occurs only at the site at which phosphatase is present in the tissue and only if a pH suitable for this reaction (Ca. 9) is maintained. This is accomplished by incubation in a solution of sodium barbital.

3. In the presence of calcium salts (e.g., calcium nitrate) phosphate ions, as soon as released, combine with calcium ions to form calcium phosphate. The latter salt is insoluble in the medium used and, therefore, precipitated at the site of its formation.

4. In order to make the precipitated calcium phosphate visible in the section it is stained as recommended by Gomori or by Kossa's silver method.

Since with this method phosphatase is demonstrated only indirectly by producing calcium phosphate which then can be made visible by staining, it has to be shown in control sections that no precipitated calcium phosphate is present before conditions favorable for the action of phosphatase are established. This has been done by staining a number of sections of the same tissue by Gomori's or by Kossa's method without incubation.

RESULTS

Tissue was obtained for sectioning from patients with gingivae ranging in condition from relatively normal to extreme inflammation. Phosphatase was found in the tissue from every patient, as evidenced by deposits of calcium phosphate stained black by the methods described. These deposits are uniformly found within the endothelial cells of capillaries (*fig. 1*). The calcium phosphate deposited in the wall of a capillary apparently fills all endothelial cells of the vessel.

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incubated for 2 hours at 37°C. in the following mixture: 2 per cent solution of glycerophosphate (3 parts); 2 per cent solution of sodium barbital (3 parts); 2 per cent solution of calcium nitrate (1 part); distilled water (6 parts).

12. After incubation sections are washed in a dilute solution of calcium nitrate.

13. Into a 2 per cent cobaltous nitrate solution for 5 minutes.

14. Washed thoroughly in water.

15. Into a dilute solution of ammonium polysulphide for 2 minutes.

16. Washed. The phosphatase thus stained appears black. The sections are counterstained with either eosin or haematoxylin and mounted on glass slides.

The cells containing the phosphate salts are stained a deep black. The pericapillary cells appear to be devoid of phosphate (*fig. 2*). The only additional structures which occasionally are found to contain precipitated calcium phosphate are connective tissue fibers (*figs. 3* and *4*). Although the nuclei of various tissue cells appear dark in the micro-

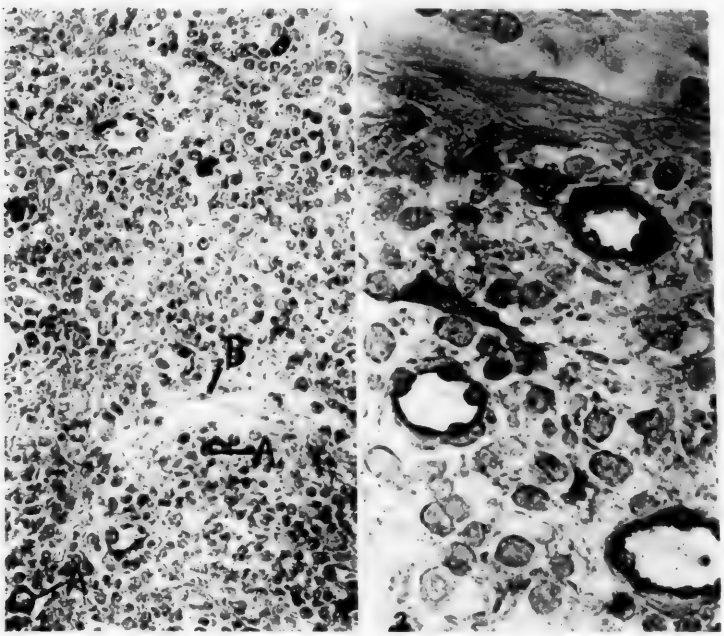

FIG. 1. Section of gingival tissue; several capillaries (*A*) containing precipitated calcium phosphate appear dark. Other capillaries (*B*) are free from this deposit. (Orig. mag. 420×.)

FIG. 2. High magnification (Orig. 1100×) of capillaries of gingivae. Endothelium of these vessels is stained deeply black indicating presence of calcium phosphate, while pericapillary tissue is devoid of it.

photographs, they are actually stained blue and, therefore, do not contain phosphatase.

Neither in the epithelium forming the outer layers of gingival tissue nor in any constituent of gingiva other than those mentioned is phosphatase demonstrable by the above method. It is reasonably certain

that epithelial cells, whether covering gingival tissue or when desquamated in the saliva, do not contain this enzyme. In order to demonstrate the correctness of the last statement, examination has been made for phosphatase by the described method of 6 smears of saliva from different individuals. In no instance has it been possible to

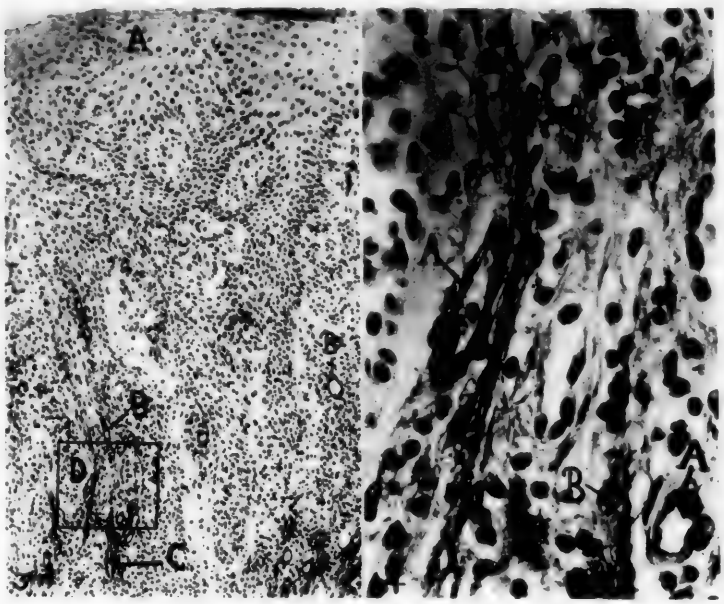

FIG. 3. Section of gingival tissue (Orig. mag. 130×). *A*. Epithelium, free of calcium phosphate deposits. *B*. Capillaries containing calcium phosphate. *C*. Connective tissue fibers containing calcium phosphate. *D*. Area shown in *fig. 4*.

FIG. 4. Magnification (Orig. 1100×) of area (*D*) from *fig. 3*. *A*. Capillary containing calcium phosphate. *B*. Connective tissue fibers containing calcium phosphate.

demonstrate even traces of the enzyme in the desquamated epithelial cells in such saliva.

DISCUSSION

The results of this bio-histologic investigation of gingival tissue have shown that phosphatase is present only in capillary walls and, perhaps, in some collagenous fibers. No phosphatase was found in the epithe-

lium. Because of this, the results of chemical determinations may be expected to vary greatly with different methods of biopsy. Thus a long superficial cut will produce a sample of tissue containing a relatively large amount of epithelial cells, while the reverse would hold true for a section consisting largely of fibrous connective tissue. In addition, the height of the epithelium and the vascularity of the tissue varies in different individuals and in certain disorders.

Smith's theory of calculus formation, holding the supposed phosphatase content of desquamated epithelial cells in saliva responsible for the phenomenon, has been discussed by other authors [Bibby (7), Rosebury and Karshan (8)], who found theoretical reasons for doubting the validity of his conclusions. The results of the present study have shown that epithelial cells, whether part of the superficial layers of tissue or desquamated in saliva, are devoid of phosphatase. Therefore, it is doubtful whether the variation in the amount of desquamated cells in saliva is responsible for variation in the phosphatase content. The variations in phosphatase content which Smith found were possibly due to the fact that he used chloroform in order to eliminate bacterial action. Chloroform causes disintegration of bacterial cells and might liberate intracellular phosphatase which is present in certain bacteria in significant amounts. The enzyme thus released diffuses into the saliva and may have brought about the differences observed by Smith. Smith observed that centrifuging removes the phosphatase content of saliva and interpreted this result as being due to removal of desquamated cells. The result might be more readily explained by the fact that centrifuging also removed the bacteria which actually contained the enzyme.

Studies of the enzyme activities of cells and bacteria are of more than academic interest. When Robison elucidated the role of phosphatase in bone formation, he also provided a possible explanation of similar processes observed at other sites in the body. The mechanism of transforming dissolved mineral salts into insoluble deposits is said to be essentially identical in bone and calculus formation. The action of phosphatase may also explain further details of calculus formation as described by Naeslund (9), who decalcified salivary calculus and showed that the framework of calculus consists of a network of bacteria such as actinomyces, leptothrix and streptothrix. It is not

unlikely that significant amounts of phosphatase could be isolated from cultures of such bacteria.

Normally the phosphatase of the subepithelial tissue is prevented from reaching the saliva by the layers of epithelial cells. Any amounts of enzyme present in the saliva of normal individuals can, therefore, be derived only from the oral bacteria. They may be responsible for the formation of some of the salivary calculus. However, in cases of gingival inflammation the formation of pockets with destruction of the epithelial lining exposes subepithelial tissue having capillaries containing phosphatase. The serum which exudes from the denuded tissues may contain the materials which result in the formation of calculus.

SUMMARY

1. The role of phosphatase in the deposition of calcium phosphate is discussed.

2. The presence of phosphatase in the endothelial cells of the capillaries of the subepithelial gingival tissues has been demonstrated by bio-histologic methods.

3. The epithelium of gingival tissue was found to be devoid of phosphatase.

4. A small number of connective tissue fibers of the gingivae appeared to contain phosphatase.

5. The significance of these findings in relation to calculus formation is discussed.

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ADAPTATION AND TISSUE TOLERANCE OF SILVER ROOT CANAL FILLINGS[1]

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It has been rather generally agreed, for some time, that the treatment of pulp-involved teeth is good practice under certain circumstances. A number of men have demonstrated the deposition of new cementum in the apical foramina of *properly* treated pulpless teeth. Others have shown, by serial radiographs taken over a period of years, that repair of damaged periapical tissue follows treatment, and that such areas remain healthy.

ESSENTIALS FOR SUCCESSFUL TREATMENT

Successful treatment is obviously dependent upon several cardinal factors—case selection, surgical cleanliness, minimum trauma, and a well-adapted filling. The last of these has taxed the skill of the best operators. If a canal is overfilled there is impingement upon the periapical tissues. They may tolerate such impingement, if it is not too great, but the process of healing is complicated and retarded when foreign substances protrude beyond the apex. On the other hand, if a filling is short—short in relation to the cleansed canal—a space is left where exudates may accumulate. Such an area is certainly one of lowered resistance, and may readily become a focus of infection. Similarly, a porous filling is hazardous. Unless a dense and well-adapted filling is inserted the purpose of the filling may be defeated, no matter how well the other phases of technique are worked out.

RELATIVE MERITS OF GUTTAPERCHA AND SILVER

Guttapercha has long been a favorite material for filling root canals, and is a good one when well condensed or closely adapted to the walls

[1] Read at the 19th General Meeting of the International Association for Dental Research, St. Louis, Mo., March 15-16, 1941. (*J. D. Res.* 20: 248, 1941.) (Received for publication April 13, 1941.)

of the canal. Coolidge (1), Kronfeld (2) and others have shown that it is readily tolerated, new cementum being deposited immediately adjacent to it. However, since guttapercha readily changes shape under

FIG. 1. A. Radiograph made at time of placement of silver point (2/15/40). B. Radiograph of same tooth made 12/13/40.

FIG. 2. A. Radiograph made several days after pulp removal and canal filling (6/2/37). Note thickened periodontal membrane. B. The periodontal membrane has resumed normal appearance. Silver cone in distal root protrudes slightly beyond apex but adjacent tissue shows no evidence of inflammation, radiographically (5/16/38).

pressure, it is often very difficult to prevent overfilling during the condensing process. Incidentally, it is extremely tedious to fill the fine canals of multi-rooted teeth with guttapercha.

The desire to simplify this important phase of root-canal therapy prompted the author (3), in 1931, to have silver cones made which

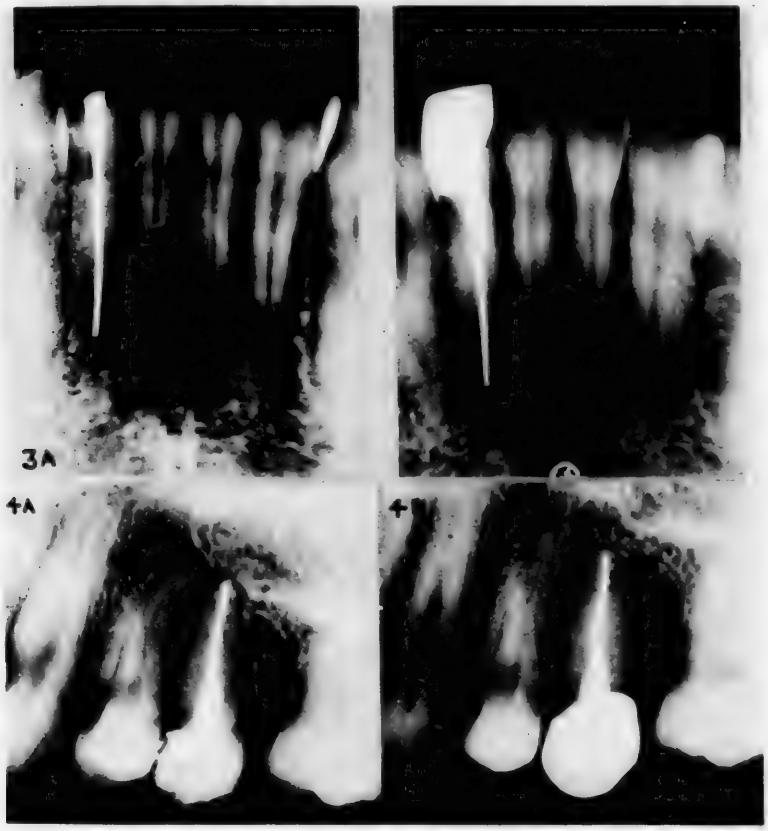

FIG. 3. A. Filling material protrudes somewhat beyond apical foramen (3/1/35). B. It is apparently well tolerated as the bone surrounding silver cone appears same as that about adjacent teeth (12/14/40).

FIG. 4. A. Radiograph made immediately after canal filling (12/3/34). B. Six years later (11/9/40) root appears to be longer than root canal filling, probably as result of new cementum.

correspond to standard instruments. The fact that root canals present many irregularities, and are rarely round, was not overlooked. Of necessity canals must be enlarged so that they can be cleansed. Since

this enlarging is done with round, tapered instruments, at least the apical portion of the root canal is made round and definitely tapered. These cones conform to such apertures. When a cone has been fitted, it is sealed in position with a bland compound such as that suggested by Dixon and Rickert (4). Guttapercha points are often used in the coronal two-thirds of the canal to more securely wedge the silver cone, but this is simply a mechanical detail.

In suggesting these cones, the author was aware that silver root-canal points had been advocated by others. However, the desirability of having them made to conform to the proportions of standard canal-enlarging instruments was either not recognized, or was considered beyond the available manufacturing facilities. If a cone fits the

TABLE I

Success of treatment and filling with silver points in 100 cases observed radiographically

	CLINIC	OFFICE	TOTAL
Cases treated	79	21	100
Pulp extirpations	36	9	45
Successfully treated	30	8	38
Infected teeth	43	12	55
Successfully treated	33	10	43

canal loosely, or makes contact only where irregularities exist, too much reliance must be placed on the sealing compound. Shrinkage or porosity may readily occur.

TISSUE TOLERANCE

In order to determine the reaction of the periapical tissues to this type of filling, it was endeavored to periodically recheck patients treated in the clinic. Contact was lost with many, but it has been possible to follow approximately 80 individuals for periods ranging from 2 to 7 years. This group together with 21 from the author's private practice, constitutes 100. Of this number, 81 per cent shows apparently perfect tissue tolerance, radiographically (Table I).

In the grossly infected cases, as evidenced by considerable periapical bone loss, it was easy to follow regeneration. In cases of vital pulp

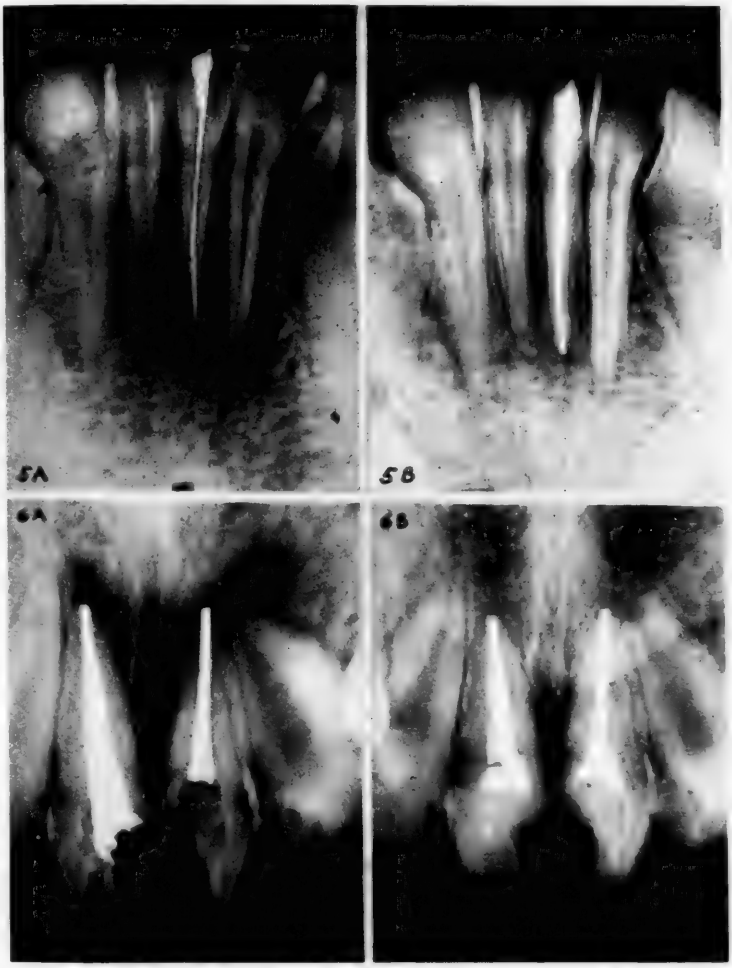

FIG. 5. A. Radiograph taken 2/2/38. B. Complete regeneration of bone following control of periapical infection (1/25/39) is another illustration of tissue tolerance of this type of filling material.

FIG. 6. Saving neglected teeth of children is one of the most important objectives of root canal therapy. Large apical openings often found in these cases are much more readily occluded by this method. A. Radiograph made 10 12 39. B. Radiograph made 7/1/40.

extirpation, it was sometimes difficult to note periapical reactions. However, a thickening of the peridental membrane was almost invariably observed several days after pulp removal and canal filling, followed by a gradual return to normal. Only those cases in which the peridental membrane resumed its normal appearance were considered successful (*figs. 1–6*).

CONCLUSIONS

Since the periapical tissues adjacent to 81 per cent of the teeth examined showed either complete regeneration following treatment, or only temporary alteration following pulp removal, it can reasonably be assumed that the failures were due to factors other than the silver. This assumption is based on the fact that extensive surveys of guttapercha-filled root canals, made at the College of Dentistry of the University of Illinois (5), show comparable failures; in fact a slightly higher percentage.

Other factors being equal, the mechanical advantages of silver for this purpose make it the material of choice.

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EFFECTIVENESS OF CHILLED BURS IN ELIMINATING PAIN[1]

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The exact mechanism by which pain is transmitted through the dentin to the sensory fibers in the dental pulp is not known. Three theories which have been advanced to explain this phenomenon are as follows: 1. Tomes' process of the odontoblast is a protoplasmic structure capable of being irritated and therefore responsible for the conduction of impulses to the odontoblast and to the sensory nerve endings in the pulp. 2. Sensory nerve fibers exist in the dentin and conduct impulses through the dental pulp to the central nervous system. 3. Alterations of the hydrostatic pressure of the dental lymph in the dentinal tubules are transmitted to the odontoblasts, which in turn stimulate the sensory nerve endings in the dental pulp.

The application of cold to the teeth for the purpose of anesthetizing these organs during the preparation of cavities has been attempted in several ways. Among these may be mentioned the early use of snow by Thomas Bartolinus; the ethyl chloride spray; Fabret's gazotherm, and certain very volatile solutions which cause cold by evaporation.

The present study was undertaken to evaluate clinically an apparatus developed on the basis of the hydrostatic theory of pain transmission in the dentin. The inventor's idea was that if a sufficiently low temperature could be applied to the immediate area of dentin being cut at the moment, the protoplasmic fluid in the dentinal tubules would be congealed. This would allow neither movement of the fluid nor the usual resultant movement of the odontoblast which he claims stimulates the nerve endings in the pulp. To accomplish his purpose, the following apparatus was developed:

1. An upright carrier for a 20 pound tank of liquid carbon dioxide.

[1] Read at the 19th General Meeting, International Association for Dental Research, St. Louis, Missouri, March 15–16, 1941. (*J. D. Res.* **20**: 242, 1941.) (Received for publication March 31, 1941.)

2. A pressure indicator attached to the tank. 3. Flexible spiral metal tubing leading from the indicator to—4. A special handpiece containing an expansion chamber around the base of the dental bur. An exhaust tube allows escape of the carbon dioxide gas at the base of the handpiece. A bakelite sleeve covers the handpiece, protecting the operator's hand from the intense cold which develops within the handpiece.

The technic for using this apparatus is as follows: 1. Isolate the tooth by the application of the rubber dam or the use of cotton rolls. 2. Drill into the tooth with a bur (not chilled) until the patient feels pain. 3. Chill the bur by opening the valve on the tank of carbon dioxide. This allows the liquid carbon dioxide to flow through the fine spiral tubing into the handpiece and expand there with the production of intense cold around the base of the dental bur. While being chilled the bur is revolved slowly, not in contact with the tooth. This chilling requires approximately 30 seconds. 4. Apply the chilled bur, not revolving, to the cavity. 5. After a few seconds, start the bur revolving slowly. Maintain a constant slight pressure and drill continuously until the drilling is completed. The carbon dioxide is left turned on as long as drilling continues. If necessary to move the bur to another part of the cavity, the first contact should again be made with the bur not revolving. After the chilled bur had been used, the unchilled bur was again applied in the cavity to further compare the effectiveness of the two burs.

The patients included in this study were carefully selected in order to eliminate in so far as possible the psychological effect which such an apparatus might produce. The patients were classified as being phlegmatic, average, or hypersensitive. The study included no phlegmatic patients, 26 average patients and 7 hypersensitive patients. The teeth operated were also classified as being hyposensitive, average, or hypersensitive when tested with the regular unchilled bur. There were 7 hyposensitive, 31 average and 5 hypersensitive teeth in the 43 teeth operated. It was not possible to show any relationship between the classification of patient and sensitivity of the teeth.

This clinical study was carried out on 43 teeth from 33 patients, 20 females, 13 males, ranging in age from 17 years to 61 years. The distribution of the teeth in which cavities were prepared was: max. 1st

incisors, right—2, left—1; max. 2nd incisors, right—3, left—2; max. canines, right—3, left—2; max. 1st premolars, right—3, left—2; max. 2nd premolars, right—2, left—1; max. 2nd molar, right—1; mand. 1st incisors, right—1; mand. canines, right—5; left—2; mand. 1st premolars, right—4, left—2; mand. 2nd premolars, right—2, left—2; mand. 1st molar, right—1. Thirty-eight of the 43 cavities were prepared in the cervical region of the crown, admittedly one of the most sensitive areas of the entire tooth.

TABLE I

Comparison of chilled and unchilled burs

NUMBER TEETH	EFFECT OF CHILLED BUR ON PAIN							
	Eliminated		Decreased		Unaffected		Increased	
	No.	Per cent	No.	Per cent	No.	Per cent	No.	Per cent
43	6	14	27	62.8	7	16.3	3	7.0

TABLE II

Teeth with painful reaction to first contact of chilled bur

FIRST CONTACT OF CHILLED BUR AND DENTIN			
Painful		Not painful	
No.	Per cent	No.	Per cent
24	55.8	19	44.2

The results of the study are shown in Tables I and II. Six teeth (14 per cent) were drilled painlessly when the chilled bur was used. Twenty-seven teeth (62.8 per cent) were less painful with the chilled bur than with the regular uncooled bur. In 7 teeth (16.3 per cent) there was no appreciable difference in pain whether the bur was chilled or not. Three teeth (7 per cent) reacted more painfully when the chilled bur was used. Thus in 76.8 per cent of the 43 teeth operated the chilled bur reduced or eliminated the pain. Twenty-four teeth (55.8 per cent) responded with sharp pain when the chilled bur was first applied to the cut surface of the dentin.

To determine how much the cold bur reduced the temperature of the area of dentin with which it was in contact while operating, a standard

electrical potentiometer with a copper-constantan thermocouple was employed. Temperature determinations were made in vivo on 4 teeth from 4 male patients. The thermocouple was embedded in the dentin and held in position by means of oxyphosphate of zinc cement, as seen in Table III. This figure shows the area of the tooth cut by the burs during the experiment. It will be noted that the chilled bur operated at a temperature of from 10° to 23.1°F. lower than the bur which was not chilled. Whether this difference is sufficient to explain the results obtained remains a matter of speculation.

The possibility of pulp damage as a result of applying the chilled bur to the dentin was considered. For this study, cavities were prepared in 8 sound teeth which were to be extracted from 4 patients (2 teeth from each patient). In each patient 1 tooth was operated on with the chilled bur, and as a control, a cavity was prepared in the other tooth with the unchilled bur.

Fig. 1 is a low-power photomicrograph of an upper right first premolar from a 40 year old female. A cavity was prepared on the buccal surface with a # 558 cross-cut fissure bur which was not chilled. The preparation of the cavity caused considerable pain. The cavity was sealed with gutta-percha and varnish, and the tooth extracted 72 hours later. It will be noted that there is a small zone of hemorrhage in the pulp tissue near the odontoblasts whose fibers were cut during the cavity preparation. This tooth served as a control for the upper right second premolar from the same patient, seen in *fig. 2*. This cavity was also prepared on the buccal surface, with a chilled # 558 cross-cut fissure bur and caused only slight pain. The cavity was sealed with gutta-percha and varnish and the tooth extracted 72 hours later. Here, as in *fig. 1*, there is a small zone of hemorrhage in the pulp tissue under the cavity preparation. In neither tooth did these pulp reactions give rise to clinical symptoms.

Fig. 3 is a low power photomicrograph of a lower right second premolar from a 36 year old male. The cavity was prepared on the buccal surface using a chilled # 35 inverted cone bur. The cavity was sealed with gutta-percha and varnish and the tooth extracted $1\frac{1}{2}$ hours later. There was considerable pain during this preparation, but it is of interest to note that in this case the carbon dioxide pressure was

TABLE III

Highest temperatures (°F.) recorded by means of electrical potentiometer when using the chilled and unchilled burs

R. T. = regular temperature

CASE NUMBER		1	2	3	4
Highest Temp. (°F.)	R. T. Bur		118.6	108.8	102.0
	Chilled Bur	88.8	95.5	95.0	92.0
Difference (°F.)		?	23.1	13.8	10.0

Thermocouple embedded here.

Area operated.*

Room Temperature = 68°F.

Engine Speed = 1200 r.p.m.

* The drawing shows the area of the teeth operated for the temperature determinations.

FIG. 1. Low power photomicrograph, upper right first premolar, 40 year old female. Cavity (C) prepared with unchilled bur. D = dentin; D′ = dentinoid; O = odontoblasts; P = pulp; H = area of hemorrhage.

lower than is necessary for effective chilling of the bur. There is, after $1\frac{1}{2}$ hours, no damage evident in the pulp tissue under this cavity preparation.

FIG. 2. Upper right second premolar from same patient as *fig. 1.* Cavity prepared with chilled bur. Same legends.

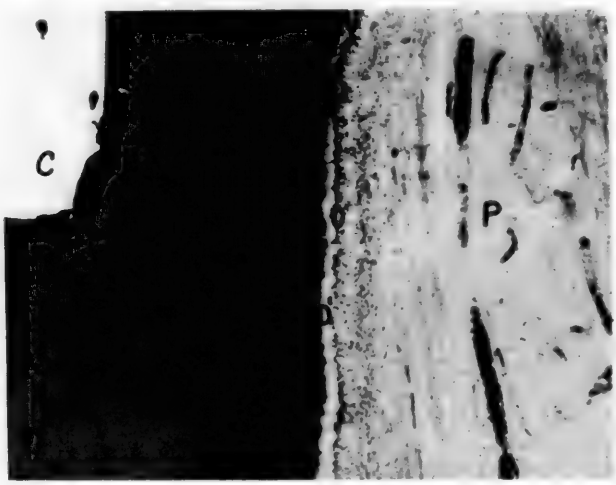

FIG. 3. Low power photomicrograph, lower right second premolar, 36 year old male Cavity prepared with chilled bur. Same legends as *fig. 1.*

SUMMARY

The results of this study indicate: 1. The use of chilled burs diminished or eliminated pain in 3 out of 4 patients (76.8 per cent). 2. The chilled bur operated at temperatures ranging from 10° to 23°F. lower than the unchilled bur. 3. The reactions seen in the pulp were of minor importance and occurred as regularly in the teeth operated with the unchilled bur as with the chilled bur.

The author wishes to express his thanks to the Divisions of Diagnosis and Operative Dentistry for their aid in securing suitable patients for this study, and to Dr. Herbert Ayers of the Division of Dental Materials for his aid in the temperature determinations.

CONCERNING THE "VITALITY" OF THE CALCIFIED DENTAL TISSUES[1]

III. Dental Lymph in the Dentin

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The recent designation that the basis substance of dentin is a "dead" tissue (1) carries with it the implication that there is no possibility of biologic activity in this tissue. In the same sense, the intercellular substance of bone is also disclosed as a "dead" tissue notwithstanding the general acceptance of its being a storehouse for calcium and that reversible processes have been demonstrated.

The division of teeth into their component parts, i.e., the pulp, enamel, dentin and cementum, has facilitated the study of these tissues. The academic value of this separation is beyond question but has its concommitant ill effect on the study of the physiologic relationship of these tissues to each other. That the possibility of biologic reactions occurring in the dentin is dependent entirely upon a vital pulp has been demonstrated by chemical and physical changes in dentin after its formation. These phenomena have been accepted as vital processes by Beust (2), Bodecker (3), and Applebaum (4). The question is not whether the basis substance possesses a form of vitality comparable to that of living cells but whether the pulp can produce chemical or physical changes affecting the calcified tissues.

Convincing evidence (5) has demonstrated the presence and diffusion of dental lymph in the dentin. The presence of the pulp established its influence by regulating the results of vital staining of the dentin. The study, made entirely by examination of ground sections, eliminated the possibility of studying the pulp. Water and alcohol soluble stains are impracticable for decalcification in vital staining. Silver

[1] Presented at the 19th General Meeting of the International Association for Dental Research, St. Louis, Missouri, March 15, 1941. (*J. D. Res.*, 20: 242, 1941.) (Received for publication April 13, 1941.)

stains, such as argyrol and silver nitrate, were found to be equally illuminating in both ground and decalcified sections. Teeth of dogs, calf and human beings were stained vitally. A small cavity, $\frac{1}{2}$ mm. in diameter, was prepared at the neck exposing the pulp. A few particles of argyrol or crystals of $AgNO_3$ were inserted into the pulp and the cavity sealed either with cement or gutta percha. The specimens were taken 2 hours to 7 days later. Permanent teeth of human beings were treated in the same manner as those in the animal ex-

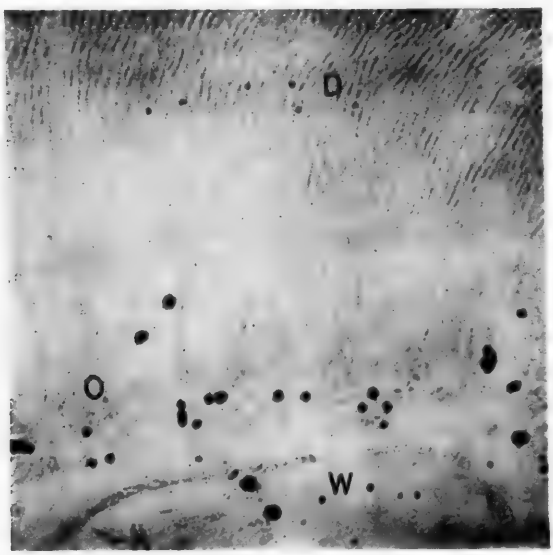

FIG. 1. Section of pulp of dog's tooth vitally stained for 2 hours with silver nitrate. Counterstained with eosin to prevent nuclear staining. Accumulation of precipitate in layer of Weil (W). Stained particles in odontoblasts (O) and dentin (D).

periments. Human deciduous teeth, which had been treated for pulp exposure by capping with red copper cement to which silver nitrate had been added (6, 7), were also utilized in this study. In all, 40 teeth of human beings and animals were examined. All specimens were decalcified either in hydrochloric, acetic or lactic acid. Sections were studied unstained, stained with eosin or hematoxylin and eosin. Attempts to study specimens stained in vivo with potassium permanganate, gentian violet and eosin were ineffectual, the stains being destroyed in the preparation of the decalcified sections.

RESULTS

This examination proved a valuable adjunct to the earlier report (5) made from the study of ground sections in so far as it was possible to study the results of vital staining in thinner sections and also to retain the pulp. In general, the results were similar in both studies.

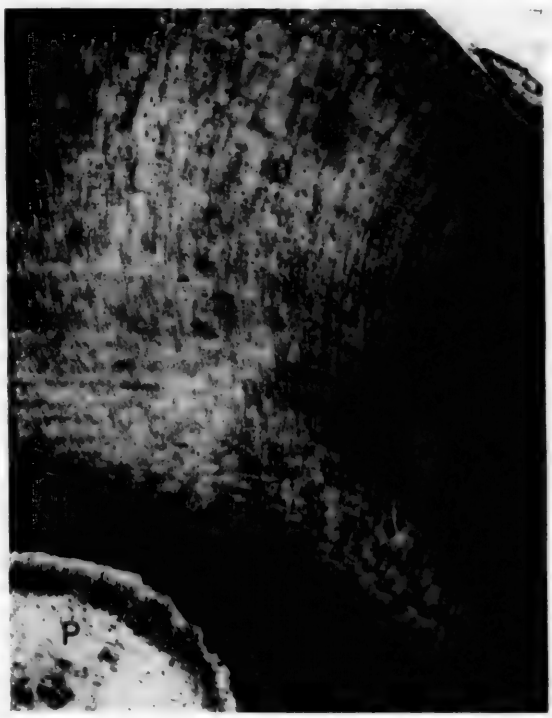

FIG. 2. Section of dentin of same specimen as *fig. 1*. Counterstained with hemotoxylin and eosin. Silver precipitate follows course of dentinal tubules (D). Stain larger than diameter of tubules and separated. Isolated tubules stained.

Fig. 1 shows a section of the pulp of a dog's tooth which had been stained in vivo with silver nitrate for 2 hours. This section counterstained with eosin shows the accumulation of precipitated silver nitrate in the layer of Weil. Several large particles may be observed in the odontoblasts while others, smaller in size, may be observed in the dentin. The profuse diffusion of the dye in the dentin may be seen in *fig. 2*, taken from the same specimen. The dye permeated the entire

body of the dentin, following the curvature of the tubules. In this area the dye failed to penetrate every tubule but was observed in selected or isolated tubules, the remainder being unaffected. This was observed only in the circumpulpar dentin. Furthermore, it may be noted that the precipitate varies in size and that the larger particles are several times the diameter of the tubules and widely separated. By contrast, the staining of the peripheral dentin in the same specimen is remarkably uniform; the particle size being approximately the same as the diameter of the tubule and closely packed. All the tubules in

FIG. 3. Interglobular dentin of dog's tooth stained in vivo with silver nitrate 2½ hours. Counterstained with hemotoxylin and eosin. Precipitate accumulates in partially calcified matrix.

the peripheral dentin are stained demonstrating the so-called "lymph plexus" of Fish (8).

Previous description of dental lymph failed to show any evidence of the stain particles within the basis substance of the dentin. The passage of the dye beyond the tubules was noted in the interglobular dentin of a dog's tooth which had been vitally stained for 2½ hours. In *fig. 3* can be seen the passage of stain particles beyond the tubules where they accumulated in the uncalcified matrix. *Fig. 4* is a section of a human deciduous tooth which has been treated for pulp exposure as previously described. The tubules, cut longitudinally exhibit the silver precipitate within Tomes fibers and lateral branches. *Fig. 5*

presents further evidence of the passage of dental lymph into the basis substance. This is a section of dentin of a sheep which had been stained in vivo for 4 days with argyrol. The resultant sections suggest that silver salts in solution had passed into the basis substance and were precipitated during fixation with formalin. Similar results

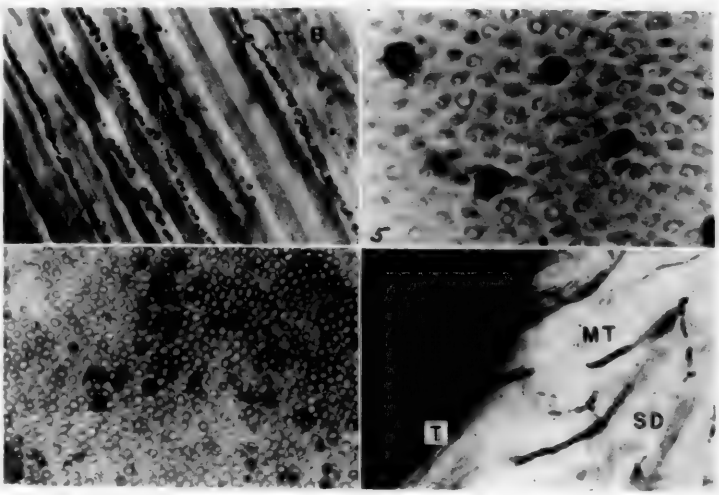

FIG. 4. Section of human deciduous incisor treated for pulp exposure with red copper cement and silver nitrate. Precipitate confined within Tomes fibres and extended into lateral branches (LB).

FIG. 5. Section of dentin of sheep vitally stained for 4 days with argyrol, counterstained with hemotoxylin and eosin showing transverse section of dentinal tubules. Large size of precipitate suggests passage of stained dental lymph into basis substance.

FIG. 6. Transverse section of dentinal tubules of dog's tooth stained 24 hours with argyrol. Fibrillar staining—F. Perifibrillar staining—P.

FIG. 7. Ground section of sheep's tooth stained with silver nitrate in stain pocket in dentin. Centripetal staining in tubules (T) which penetrated to pulp. Metamorphosed tubules (MT) cut off from communication with pulp and secondary dentin. Centrifugal staining in tubules of secondary dentin (SD).

may be observed in the large stain particles in *fig. 2* in longitudinal sections of the tubules.

Transverse sections of dentinal tubules give evidence of 2 possible modes of passage. *Fig. 6*, a section of a dog's tooth 1 day after insertion of the stain in the pulp, shows the presence of the stain in Tomes fibers which appear tubular and do not occupy the entire lumen

of the tubule. The fibers vary in diameter and as in the other sections not every tubule is stained. In the same field, however, staining of the perifibrillar space may be observed. Further evidence of a two-way flow may be seen in the tooth of a sheep in which silver nitrate had been deposited in a stain pocket in the dentin (*fig. 7*) for 4 days. In this section the diffusion toward the pulp had been arrested at the border of secondary dentin. Staining of the pulp occurred through a strand of uninterrupted tubules. The centrifugal flow can be seen in the staining of tubules of the secondary dentin arising from the pulp while the centripetal flow is demonstrated in those tubules which transported the dye to the pulp from the stain pocket.

DISCUSSION

The observations of the diffusion of stains from the pulp through the dentin add further evidence to the previous studies of dental lymph. The present study suggests that the silver salts in solution passed into the odontoblasts, through Tomes fiber into the basis substance of the dentin and were precipitated by formalin during fixation. The dye permeated the entire pulp and concentrated in the layer of Weil. In the dentin, the diffusion was observed only in those areas where the odontoblasts were normal. In many specimens, areas of degenerative changes in the odontoblastic layer were present. It is interesting to note that the dentin in these areas was not stained suggesting that these cells play a role in the distribution of dental lymph. A similar failure of dye penetration was noted in metamorphosed dentin in ground sections (5).

The passage of dental lymph transporting dissolved silver salts into the basis substance suggests that it is not confined to the tubules alone but diffuses into the basis substance. The failure to make this observation in the previous report (5) was due to the thickness of the ground sections and the inability to examine transverse sections of the tubules.

The presence of a two-way flow to and from the pulp is again demonstrated (5). The observation of a centrifugal and centripetal flow of dental lymph justifies the working hypothesis of Bodecker (9) which suggested a two-way flow through the dentinal tubules.

Several observations made in this study cannot be explained. The

failure of the dye to penetrate every tubule in the circumpulpar dentin and profuse staining of the peripheral area remain problems for further study. Likewise, variations in the size and the complete absence of Tomes fibers is still to be explained.

The dental lymph present is not without a function. Attempts to explain the protective metamorphosis of the dentin, the presence of a translucent zone under caries as well as the phenomena of posteruptive calcification could not be attributed to a "dead" tissue. The important consideration is that the vital processes of the dentin are dependent on its formative and nutrient organ, the pulp. Physiological responses of dentin can only occur in so far as a vital pulp is present. One function of the pulp is to provide the basis substance with its vital tissue fluid, the dental lymph. Up to the present status of the physiological study of dentin, the flow of dental lymph is the only known means of biologic changes in the dentin.

The evidence of radioactive phosphorus and fluorin in dentin after intravenous injection or mouth feeding (10, 11, 12, 13, 14) has been subjected to some criticism on the basis of absorption from the saliva. The mechanism of passage of silver solutions is undoubtedly similar to that of radioactive phosphorus.

Recent trends in dental research regard dentin as a "dead" tissue; as such, it would be deprived of the benefits of biological reaction to irritants and disease. If this be true, it must be considered a foreign body. This is untenable in the light of the presence of dental lymph which through the odontoblast is in communication with the general circulation.

SUMMARY

Dry dyes placed in the pulp diffused in 2 directions through the dentin. The flow is dependent on a normal odontoblastic layer. Dental lymph in communication with the general circulation through the pulp establishes assuredly a low degree of vitality of dentin. The mechanism of the afferent and efferent flow is suggested as well as the diffusion of dental lymph into the basis substance.

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CONCERNING THE "VITALITY" OF THE CALCIFIED DENTAL TISSUES[1]

IV. The Vital Staining of Human Dental Enamel

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A knowledge of the permeability of dental enamel is important in the comprehension of the possible physiological activity of the calcified dental tissues. It is noteworthy that this permeability is highest in the lower forms of Mammals and lowest in Man.

Permeability of teeth can be demonstrated *in vivo* by dyes and by the administration of radio-active phosphorus. The former method is the older as used by Prinz (1), Fish (2) and Bertram (3), and, even though it may be more gross, it has the advantage of identifying the permeable structures and establishing their exact location. Investigations which combine the two methods can be expected to give highly informative data concerning the diffusion of body fluids through enamel, dentin and cementum.

The vital staining method has been used by W. Lefkowitz (4), to date, on 183 teeth to determine the permeability of the calcified dental tissues in dogs, sheep, calves, monkeys and man. It has been observed that the permeability of the enamel, dentin and cementum is in direct relation to their organic content. Enamel having the least organic material, is much less permeable than dentin.

Dental enamel of marsupials appears to be the most permeable of all mammals, since a great number of tubules pass into it from the dentin. The tubular character of the enamel in the opossum (Didelphis virginiana) is clearly visible (*fig. 1*). These animals are a remnant of a prehistoric group of mammals. As we progress to the more recent species, the permeability of the enamel is somewhat reduced.

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The ungulates and carnivores still have quite permeable enamel, but tubules similar to those found in marsupial enamel cannot be recognized. The teeth of cows and horses show enamel with highly permeable structures such as tufts, spindles and lamellae. These, as well as the rod sheaths and cement substance are stainable. Decalcification demonstrates a considerable organic residue in dental enamel of a horse (*fig. 2*).

Berke (5) by means of a careful method of decalcification observed that the bands of Schreger in enamel alternate in areas of high and

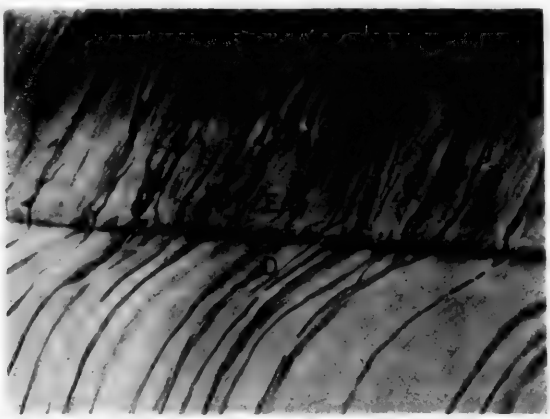

FIG. 1. Ground section of molar of opossum showing tubular character of enamel (E). D = dentin.

low content of organic matter. These structures are developed particularly well in the teeth of dogs and can be stained vitally by placing a dye in the pulp chamber.

The permeability to dyes of enamel and dentin, besides being dependent on the amount of their organic content, is affected also by their maturity. Teeth which have been in the mouth for a period of 5, 10 or more years have a greatly reduced permeability. The cement substance between the enamel rods, the tufts and lamellae are permeable during youth. However, later most of these channels become blocked in human teeth. The dentin, likewise, loses much of its permeability by blocking of many of its tubules (6). Consequently, if a dye is placed in the pulp it will pass through the dentin

and penetrate the enamel to a greater depth through the open channels of a young tooth than through the blocked ones of a matured tooth.

A previous publication (4) has dealt with the more gross distribution of dyes in the enamel and dentin as a result of vital staining. This paper will show some of the more minute channels in enamel through which the dental lymph transports dyes.

W. Lefkowitz (4) stained teeth of dogs vitally by sealing various dyes in the pulp for from 2 hours to 7 days. The agents used for the vital staining experiments were gentian violet, eosin, methylene blue, Sudan IV, silver nitrate, argyrol, safranin and osmic acid. Silver

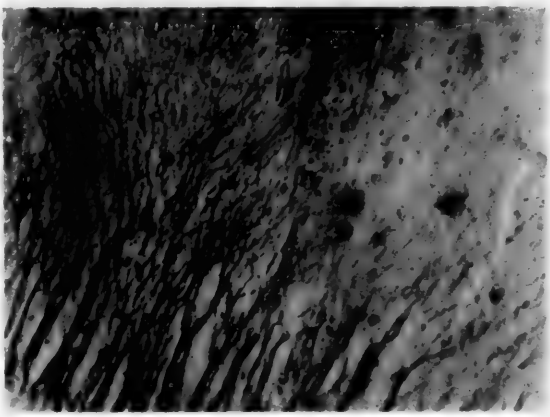

FIG. 2. Enamel of horse incisor decalcified under coverglass showing high content of organic matrix. (Specimen could not be cleaned without derangement of structure.)

derivatives were found to be the most satisfactory both for the purpose of photography and their stability during the decalcification of the experimental teeth. Teeth of dogs have been observed to be more permeable than those of adult human beings (4). *Fig. 3*, a decalcified section of a dog's canine, shows the silver nitrate particles clearly in the dentin as well as in the residual enamel matrix.

The enamel of deciduous teeth of children also shows a definite permeability. This is demonstrated by the microscopic examination of teeth in which the pulps had been capped. Stowe (7) and Rosenstein (8) have been using a silver nitrate cement for this purpose with a high degree of success. After the placing of the cement it is evident

that a certain amount of silver nitrate is dissolved in the pulp and carried through the adjacent dentin into the enamel (*fig. 4*). Only a small amount of enamel matrix remains after decalcification in which

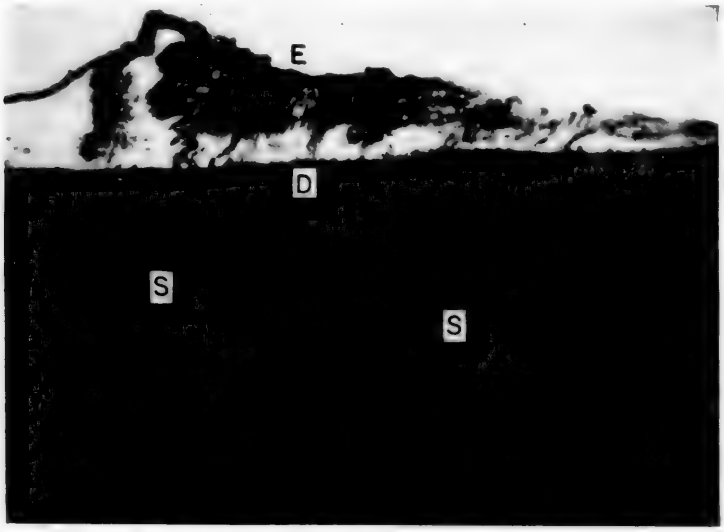

FIG. 3. Decalcified section of canine of dog showing vital staining (S) of dentin (D) and enamel matrix (E). Dentinal tubules and enamel rod sheaths carry silver nitrate originally placed on vital pulp.

FIG. 4. Deciduous, human tooth stained vitally by placing silver nitrate on pulp. Stain granules visible in dentin (D) and enamel matrix (E).

FIG. 5. Same as fig. 4. Silver nitrate granules outline a tuft (T).

the silver nitrate crystals can be seen. A tuft is noted in which the stain granules clearly outline this structure (*fig. 5*). The fact that vitally stained enamel shows the dye localized particularly near the

dentin, indicates that its origin is from the pulp. Experiments with radio-active phosphorus show only the presence of the phosphorus but do not localize it to any specific *structure* of the enamel.

The question whether the enamel is permeated solely by the dental lymph or solely by the saliva or by both of these fluids has not been settled. The recent observations of Sognnaes and Volker (9) show that saliva penetrates the enamel of teeth of monkeys. This evidence was presented on the basis of observations of the presence of radioactive phosphorus in the enamel. It appears that they set up their experiments with the intention of determining not only whether radioactive phosphorus could be found in thee namel but also whether it originated from the saliva or from the dental pulp. For this purpose certain teeth were covered with caps and it was observed that the enamel of these contained radioactive phosphorus near the dentin. The teeth without caps, on the other hand, showed a greater quantity. This proves that the saliva was the carrier of the greater part of the phosphorus found in the teeth. However, the question arises: did the reagent pass through the untouched *surface* of the enamel or did it penetrate the enamel worn by the stress of mastication?

It would be interesting to continue the experiments with radioactive phosphorus on recently erupted teeth, having intact occlusal surfaces The reason for suggesting further investigation is that teeth which have been worn extensively by mastication have a greatly reduced permeability, particularly from within. The irritation caused by the exposure of the dentin results in the formation of considerable secondary dentin. This structure cuts off the primary dentin from the pulp greatly reducing the diffusion of the dental lymph. The old conception that the physical condition of teeth remains the same, whatever their age or degree of attrition, may be responsible for some erroneous generalizations concerning possible physiological activity in the calcified dental tissues. The experiments of W. Lefkowitz (4) indicate that the *normal* enamel of *matured*, human teeth in the mouth, are impermeable to dyes dissolved in saliva. The word "normal" must be emphasized particularly, because incipient carious lesions showed a definite stain penetration, an indication that such areas were partially decalcified. The fact that the use of Andresen's mineralization powder (11) can occasionally re-calcify "white spots" in enamel is evidence that such areas can be penetrated by the saliva.

The experiments of Volker show that the saliva is charged with radioactive phosphorus and that it finds its way into the dental enamel. The fact, however, that some of the reagent was found in the enamel of teeth covered by caps and that it was present near the dentin indicates that the pulp is a source of mineral salts. The placing of caps on teeth to isolate them from the saliva may retard the diffusion of the dental lymph. This might explain the reduced quantity of radioactive phosphorus found in these capped teeth.

An outstanding observation of the present investigation is that the impermeable membrane described by Kanner (12) as separating the enamel from the dentin is non-existent; dyes passed across this junction in the teeth of dogs and children show the incorrectness of this conception. Such an impermeable barrier placed between an epithelial structure (enamel) and the underlying connective tissue (dentin) is found *nowhere* else in the body. Throughout the body, the epithelium which has no blood supply of its own is nourished by the underlying connective tissue.

The similarity which exists between epithelium and enamel is as follows: 1. both are of ectodermic origin; 2. neither have their own blood supply; 3. epithelium is nourished by body lymph diffusing through the intercellular clefts; 4. enamel has a structure similar to epithelium; the rods are held together by a cement substance which is permeable during youth as shown by present experiments.

The principal difference is that epithelium is a soft tissue while enamel is a calcified tissue.

There is no doubt that epithelium requires nourishment. If this ceases, it degenerates and sloughs off *en masse*. Does enamel likewise require "nourishment?" The answer to this question depends on the following: Is enamel of a recently erupted tooth in the same physical condition as that of a matured one? The answer appears to be "no." Karlström (13) found hardness differences between the latter and the former. Vital staining experiments show that mature enamel is less permeable than young enamel. These observations suggest that a change occurs after the eruption of the tooth and it is suggested that this change is effected by the dental lymph. A clinical proof of this statement is the change that occurs in dental enamel of *young* teeth after pulp removal. The result is an increased brittleness.

Information concerning the nature of the products furnished to the dentin and enamel by the dental lymph is mostly in the realm of speculation. Present knowledge suggests that mineral salts and a protein are present. The composition of the dental lymph is not identical with the blood necessary for the nutrition of soft tissues; it may approach the nutrient fluids of bone. Hence the terms "nutrition" and "vitality" when used in connection with the calcified dental structures may be objectionable to some investigators. The sense in which nutrition has been used by the writer is the activity of any product or products necessary for the full maintenance of function. The physiological changes which occur in enamel after the eruption of the teeth seem to warrant their being designated as resulting from "nutrition" in a limited sense.

SUMMARY

Evidence on the basis of the vital staining of teeth shows that: 1. the dentin is permeated by a fluid termed the dental lymph originating from the dental pulp; 2. the dental lymph passes not only through the dentinal tubules but also into the enamel by way of the tufts, lamellae, rod sheaths and cement substance; 3. the depth of penetration into the enamel depends on the age of the tooth. The enamel of a recently erupted tooth is far more permeable than that of a matured tooth; 4. one of the oldest of living mammals, the marsupial, has a highly permeable enamel. The enamel of man, on the other hand, has the lowest permeability.

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INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH

SCIENTIFIC PROCEEDINGS OF THE NEW YORK SECTION

June 4, 1941[1]

Compiled by FRANCES KRASNOW, Ph.D., Editor, New York Section

1. PULP STONES AND HYPERCEMENTOSIS IN ARTHRITIS. *Sidney Sorrin, D.D.S., College of Dentistry, New York University and Montefiore Hospital, New York, N. Y.* The purpose of this study is to determine whether any relationship exists between either pulp stones or hypercementosis and arthritis. Of 200 cases of arthritis studied at Montefiore Hospital, 100 were edentulous. In this edentulous group, the results of prior extractions of all the teeth were disappointing, for, the arthritis still persisted. Only those cases which had at least 15 teeth distributed between the upper and lower jaws were included in the study. Forty-six of the remaining 100 arthritics met these requirements. Thirty-seven cases, the greatest number, belonged in the chronic infectious group; 6 were found in the degenerative and 3 in the specific groups. A control group of 48 cases of so-called healthy individuals (non-arthritic) was selected at random from the Periodontia Clinic of the College of Dentistry, New York University. Only those cases which presented at least 15 teeth distributed between the upper and lower jaws were considered. Statistical study indicates no relationship between pulp stones or hypercementosis and arthritis.

2. INFLUENCE OF DENTAL DEVELOPMENTAL STATE ON PREMOLAR POSITION AFTER LOSS OF PERMANENT FIRST MOLARS. *J. A. Salzmann, D.D.S., Dental Service, New York City Vocational Schools.* Position of eruption of second premolar teeth is influenced by the dental developmental state of the child at the time the first molar is lost. When the permanent first molar is lost prior to the loss of the deciduous second molar and eruption of the second premolar, the position of eruption of

[1] The cost of publication of these sectional proceedings is paid by the New York Section. (Received for publication June 12, 1941.)

the second premolar may be markedly changed, the tooth erupting in the first molar space. The effect on occlusion of the teeth varies with the position of eruption.

3. SYSTEMIC ASPECTS OF PRECOCIOUS ADVANCED ALVEOLAR BONE DESTRUCTION: Preliminary Report. *Samuel Charles Miller, D.D.S., William Wolf, M.D., M.S., Ph.D.,* and Benjamin B. Seidler, A.B., D.D.S.,* College of Dentistry, New York University, New York, N. Y.* Local factors being insufficient to account for the widespread destruction of soft tissue and bone, 35 cases of advanced alveoloclasia in young individuals ranging in age from 14 to 30 years were studied to determine a systemic relation. Females predominated—the proportion being about 10:1. Oral radiograms of these patients were characteristic. Extensive alveoloclasia was seen chiefly in the incisor and first molar regions, both maxillary and mandibular. Radiograms of the skeletal bones revealed no discernible aberration from normal. The onset of the disease appears to occur at puberty, or in males, a trifle later. The laboratory findings for the group were: Basal metabolic rate, 6.1 per cent; Specific dynamic action, 6.7 per cent; Serum calcium, 10.7 mgs. per cent; Whole blood cholesterol, 194.0 mgs. per cent; Serum inorganic phosphorus, 2.9 mgs. per cent; Phosphatase, 2.1 Bodansky units; Glucose tolerance curve: fasting, 85.1; 1 hour, 99.1; 2 hours, 89.1; 3 hours, 85.6 mg. per cent. Although differences from the accepted normal average were not great, the trend of the group seemed to follow a definite direction. Further investigation is suggested to determine the relationship of the various laboratory figures to the oral condition, the possible existence of a negative calcium balance and the effect of the cyclic changes on the oral mucosa.

4. EPITHELIAL SMEARS OF THE ORAL MUCOSA. *Daniel E. Ziskin, D.D.S., Paul Kamen, D.D.S.,* and Irving Kittay, D.D.S.,* School of Dental and Oral Surgery, Columbia University, New York, N. Y.* The smear technic, (originally applied to investigate the cytology of the vaginal mucosa) as modified by Weinmann, was used to study the histology of the oral mucosa. The Ernst-Gram stain disclosed 6 stages of cornification, differentiated on the basis of the affinity of the cellular structures to gentian violet or safranin and the presence or absence of a nucleus.

* Guest.

The incidence of leucocytes varied inversely with the degree of keratinization. Because of the known effect of the estrogenic hormone on keratinization, tests were made to determine whether or not cyclical variation occurred which could be correlated with estrogenic levels during menstruation. Variations in oral keratinization levels during the menstrual cycle were noted, but could not be synchronized with those purported in systemic estrogen. Application of this method to the study of pregnancy, when the estrogen level falls markedly, disclosed that a low degree of keratinization and a high incidence of leucocytes exist in the normally highly keratinized palate and gingival areas. Estrin privia, lupus erythematosus, lichen planus, and leukoplakia (all associated with alterations in keratinization) were similarly examined. Variations in the smears followed the pattern of known changes in keratinization in these conditions. This technic has potential value in the diagnosis of other oral conditions.

5. THE SPIROCHETES: A NEW MOTION PICTURE. *Theodor Rosebury, D.D.S., Schools of Medicine and Dentistry, Columbia University, New York, N. Y.* In 1940 the writer, in collaboration with Mr. Paul Anderson, prepared a short film showing spirochetes under dark-field illumination. The present film is entirely new, made with improved apparatus. Accompanied by a prepared talk this film constitutes a lecture on the spirochetes, part of the course in medical bacteriology given by the Department of Bacteriology. The film includes a graphic description of the dark-field microscope, and demonstrations of preparation of material for dark-field examination from (a) a rat infected with *Treponema recurrentis;* (b) a patient with Vincent's gingivitis; (c) a guinea pig with a fuso-spirochetal abscess; (d) a culture of *T. microdentium;* and (e) a rabbit with a testicular chancre. The greater part of the film consists of dark-field photomicrographs of the following: *T. recurrentis;* the mouth spirochetes, including *T. vincenti, T. buccale, T. microdentium* and others, and bacteria of the fuso-spirochetal flora; T. pallidum and other tissue spirochetes; *L. icterohemorrhagiae* and other leptospiras; spirochete-like filaments; and *Spirillum minus.* All photomicrographs were taken at uniform magnification, so that the film shows comparative dimensions of the microorganisms as well as details of morphology and motility. Full descriptions of the cinemicrographic apparatus and of the film will be published.